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A STUDY OF
THE ARTEFACTS OF THE NERVOUS SYSTEM

The Topographical Alterations of the Gray and White Matters of the Spinal Cord caused by Autopsy Bruises, and a Consideration of Heterotopia of the Spinal Cord

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A STUDY OF THE ARTEFACTS OF THE NERVOUS SYSTEM.*

Introduction.

A knowledge of the artefacts of the central nervous system—that is, of the artificial changes produced during removal from the body and preparation for the microscope—is of a good deal of practical importance, because these changes are so very liable to be misunderstood or to be mistaken for developmental errors or for the results of disease processes.

The nervous system stands supreme over all the other organs and tissues in the high organization and extreme textural delicacy of its anatomical elements, and the complexity of their arrangements. This is why the nervous system is so much more susceptible to artificial changes, why the artefacts are so manifold and complex and deceptive in resembling ante-mortem changes in this tissue.

The firmer texture and comparative simplicity of structure of such organs as the lung or kidney permit them to endure rather rude treatment at the autopsy, without showing any extensive or perplexing artificial changes in their structures. But the nervous system demands a very different sort of treatment. The whole technique of removal and preparation for the microscope requires a much more refined and delicate procedure, and the utmost care to prevent the occurrence of artefacts. For instance, it does not seem to be appreciated that an apparently trifling compression of the fresh spinal cord, an accidental blow at the

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autopsy, or the mere careless handling of the organ, may produce changes in its complex and delicate elements and their intricate arrangement which under the microscope may suggest malformations or the results of disease processes and tempt the observer to postulate causal relationships of the artefacts to spinal-cord diseases in general, or to any particular disease which led to the removal of the cord.

As there are no works on artefacts of the nervous system, it is thought that these studies, dealing with them collectively and in detail, together with faithful drawings of their gross and microscopical appearances, may be useful as a reference paper to guard against the mistakes made about them which the literature is replete with.

A most fruitful cause of manifold artefacts in the *spinal cord* is the bruising of the organ during its removal at the autopsy. When the spinal cord is bruised—and this happens quite frequently at the autopsy—two sets of changes more or less intimately associated with each other are produced.

In the first place, the force of the blow or compression induces deformities of the gray and white matters. The two cord substances are thrust about into inappropriate places. Thus, the bruise produces (1) *topographical artefacts of the two cord substances.* Secondly, mechanical alterations in the structures of the individual elements of the gray and white substances are induced by bruises of the cord. So bruising the cord also produces (2) *minute structural artefacts in the two cord substances.*

It is with the first set of changes and the mistakes made about them that the present paper is largely concerned, while the second set of changes and other artefacts of the nervous system will be considered in a later paper.

The errors prevalent about these topographical artefacts due to bruising the cord are considerable and important. For the past ten or fifteen years observers have been describing these topographical changes in the white and gray matters of the cord, caused by autopsy bruises or manipulation, as congenital malformations, apparently without any thought of the possibility of their artificial nature, and have made it appear that the alleged malformations occur quite

frequently and induce a condition in the cord predisposing to disease. It is true that these topographical artefacts are very deceptive and do simulate malformations, and are readily produced unawares at the autopsy, but they have characteristic appearances, and can be easily traced to bruising, for a number of features plainly indicate the action of mechanical forces. Furthermore, observers should have been warned against regarding these artefacts as malformations from the fact that no peculiar symptoms or defects were ever associated with them in any of the cases.

As the subject stands to-day, some twenty-five cases of congenital malformations of the spinal cord (excluding monstrosities) are recorded in the literature, and, as a matter of fact, these cases are nothing but commonplace bruises of the fresh cord. These cases do harm by the cumulative and unchallenged, though plausible, mistakes which they perpetuate about the frequency of spinal-cord malformations and their supposed bearing on spinal-cord diseases.

On the other hand, it must be made clear that there are genuine cases of spinal-cord malformations of real developmental origin; but these are found very rarely indeed. In all, thirty-one cases of alleged spinal-cord malformations (excluding monstrosities) are recorded. *Six* of these are genuine cases. The remaining *twenty-five*, as just explained, are due to bruises or disease, or both.

Now, all of these malformations, the true and the false cases, in the adult cord are grouped together under the term heterotopia. Heterotopia (from ἕτερος, another, and τόπος, a place) means a congenital displacement of the white or gray matter. There may be a misplaced portion of the white matter, and this may be called heterotopia of the white matter. In like manner the occurrence of gray matter in inappropriate places is called heterotopia of the gray matter. The occurrence of extra portions of gray matter or fragments lying in the midst of the white matter, separated from or attached to the gray horns, is also included under the term. The term heterotopia is also applied to deformed contours of the gray matter. The word at present is used rather loosely and broadly to cover any degree of malposition of the two cord substances, from a separation of a tiny

bit of gray matter from the parent-mass up to duplication of the cord. Doubling of the cord is generally referred to as simply "duplication of the spinal cord" (*Doppelbildung*), and inequality in the volume of the gray segments is often designated as "asymmetry." (Heterotopia has also been used in much the same sense to designate little extra protuberant masses of gray matter on the surfaces of the brain cortex.) The term was first used by R. Virchow to designate misplaced fragments of gray matter in the white substance of the cerebral hemispheres.

Heterotopia of the spinal cord was first described by Arnold Pick in 1878. This was a genuine case, as were three others described by the same author in 1881. Hans Virchow mentioned a genuine instance in 1887. Thus the only genuine cases of heterotopia (excepting Virchow's case) were about the first cases published.

After these follow the erroneous cases of heterotopia due to artefacts or disease, or both. Kahler and Pick (?) in 1879, Schultze, Drummond (in England) in 1881, Fürstner and Zacher in 1882, Bramwell in 1885, and Schieffer-decker in 1887, all describe erroneous cases of spinal-cord malformations.

At this time the subject received a fresh impetus from Kronthal, who in 1890 described a congenital malformation in the spinal cord of an ox (which had been badly mangled in a butcher's store), and in 1888 recorded malformations of a cord in acute myelitis. This disease of course tends to produce secondary deformities of the gray matter, and renders the cord so soft and pulpy that the slightest manipulation causes changes in the contours and positions of the cord substances, particularly the gray matter. Kronthal's papers, which sum up the preceding cases, present statistics, and discuss the "neuropathic disposition" thought to be induced by these bruise-artefacts, were followed by quite a number of cases which extend up to the present time.

Buchholz, 1889–'90, Brasch, Jacobssohn, Feist, Turner and Campbell, and Tooth in 1891, Feist in 1892, and others describe cases of bruises as various malformations, generally under the term heterotopia, and among them two more cases associated with acute myelitis. It is surprising that such a formidable literature as this has arisen from so

simple and obvious an error without any question as to the true origin of the changes.

Seguin's case, reported by Delafield in 1872, is described very conservatively, and no conclusions are presented about its nature. Drummond is also very guarded about his case. He does not speak of it as a malformation, and seems to be the only one of these writers who had in mind the possible element of artefacts, for he states that the changes might have been due to manipulation, although he believed otherwise.

One or two rather far-reaching deductions have been made from these erroneous cases of heterotopia. One of these, the discussion of the embryological side of the evolution of these supposed malformations, does not of course invite any serious attention. But the correction of another question, of the association of the neuropathic disposition with these erroneous cases, seems to me to be of fundamental importance. Even so competent an observer as F. Schultze (and before him Kahler and Pick, but their case is very much less open to criticism) discussed at length these artificial disfigurations as an underlying basis for the neuropathic disposition. His idea was that these supposed malformations weakened the resistance of the cord against disease so as to invite the invasion of pathological processes. This association of the induction of disease with these artefacts has been reiterated from one writer to another, so that heterotopia of the cord is quite intimately associated in the literature with the neuropathic disposition. The fact is that cords the seats of actual lesions were bruised in their removal, and, under the influence of a false impression perpetuated from writer to writer, the effects of the bruising were unquestionably considered as a predisposing factor in the induction of diseases of the spinal cord.

The purpose of this paper, then, is a description of the topographical artefacts of the two cord substances incident to autopsy bruises or other manipulation, a criticism of the literature of malformations (excluding those from monsters) or heterotopia of the spinal cord, and an attempt to look at the association of the neuropathic disposition with spinal-cord malformations in its proper light.

The paper has been arranged in the following way :

Section I. A consideration of malformations of the spinal cord in general.

Section II. An analysis of the published cases of true heterotopia of the spinal cord and medulla oblongata. 1. The true cases of heterotopia of the gray matter. 2. The true cases of heterotopia of the white matter.

Section III. The methods of removing the spinal cord at autopsies and their relation to the production of artificial malformations by bruises.

Section IV. Description of the appearances of non-intentional bruises of the cord selected from ordinary routine autopsies.

Section V. A description of the results of experimental bruising of the human spinal cord. 1. Gross appearances of the bruised cords. 2. Microscopical appearances of the same. 3. Artificial duplication of the spinal cord. 4. The minute structural changes due to bruises of the spinal cord.

Section VI. An analysis of the erroneous cases of spinal-cord malformations produced by manipulation or disease, or by both combined.

Section VII. The diagnosis of spinal-cord bruises in sections, and certain general remarks.

Section I.

A CONSIDERATION OF MALFORMATIONS OF THE SPINAL CORD IN GENERAL.

Before entering into the details of the subject it will be well to consider very briefly malformations of the spinal cord in general, to see how the heterotopic condition is related to them, for the condition does really exist independent of the results of disease or manipulation.

The malformations of the spinal cord may be conveniently classed as follows:

I. Congenital Deformities associated with Monstrosities, and Incompatible with Extra-uterine Life.

These may be divided into—

1. *Amyelia*, or absence of the spinal cord. This is almost invariably associated with absence of the brain.

2. *Atelomyelia*, or partial development of the spinal cord. This is often seen in the anencephalous or acephalic monsters, where, corresponding to the incompletely devel-

oped brain, there may be various degrees of defective development of the cord as to its length; the upper portion, varying from one or two of the uppermost cervical segments to the middle dorsal region, or more, may be absent.

3. *Diastematomyelia*, a condition in which a portion or the whole of the cord is split into two lateral halves. Each half of the cord, being enveloped in its own membranes and giving rise to its own nerve roots, may fuse together to form a single cord at some region. (It is very likely that some of these cases have been confounded with an extensive degree of development of hydrorrhachis interna.)

4. *Diplomyelia*, or a formation of two spinal cords—a duplication of the spinal cord. This happens in the various kinds of double monsters.

This group of cord deformities is of no practical importance, and is only instanced here to lead up to the proper classification of heterotopia. Detailed instances of all of these grosser malformations may be found in Guerin's (1) work and the various books on teratology.

II. Minor Congenital Malformations not Inconsistent with the Maintenance of Life.

These may or may not be recognizable during life, and may be subdivided as follows:

1. *Hydrorrhachis interna*, or *hydromyelia*, a defective closure or arrangement of the divisions of the primary fœtal central canal.

This condition may be accidentally found after death. Its presence may also be indicated by the subsequent development of syringomyelia, or by its association with spina bifida, the varieties of which are well summarized in Bland Sutton's work on *Comparative Pathology*.

Under this subdivision the condition known as *hydrorrhachis externa* may be conveniently alluded to, which consists in an abnormal congenital accumulation of fluids between the meninges of the cord, causing more or less diminution in the volume of the latter.

2. Heterotopia, or misplacement of the substances of the cord.

(*a*) *There may be misplaced portions of the gray matter.* Minute bits of gray matter or of the gelatinous substance

of Rolando may develop in the white columns of the cord. These may be isolated or partially connected with the gray horns by slender gray filaments.

(*b*) *Portions of the white matter may be arranged in an unusual manner.* This malposition of bundles of white matter, or the passage of bundles of nerve fibers through unusual channels to reach their prescribed destinations, is almost exclusively observed, as would be expected, in the isthmus, where the arrangement of the white matter nearly reaches its highest complexity.

Such heterotopic conditions, however, are not indicated during life.

3. *Anomalies of the Spinal Nerve Roots.*—These are not extensive and are most marked, as a rule, in the anterior roots, where they are not infrequent. In these cases, probably, the fibers of the roots are not defective in number, but are only arranged in an unusual manner.

4. *Asymmetries of the Spinal Cord.*—Possibly some of these cases are congenital in origin to a slight extent, but it would seem that to a greater extent they are acquired during life. They will consequently be discussed under the succeeding title.

III. Malformations of the Spinal Cord acquired during Extra-uterine Life, or Secondary to Defective Development of other Parts of the Body.

These are not, properly speaking, malformations. They are produced by known pathological processes in well-developed cords and are distinct from the instances in Groups I and II. This group is not clearly defined from the congenital malformations, so that some space may be taken for their consideration as a class.

These acquired distortions of the substances of the cord may be roughly divided for our purposes into—

1. *Distortions which regularly follow some of the cord lesions.*

2. *Asymmetrical conditions of the cord due to arrested development of the cord after birth, or secondary atrophy of portions of the cord corresponding to defective development or absence of some part of the body elsewhere.*

1. The substances of the cord, especially the gray, may be distorted or misplaced by several of the spinal diseases.

It would seem unnecessary to speak of this, but these distortions, the result of certain cord diseases, have been mistaken for congenital malformations, and were supposed not only to have existed prior to the onset of the disease, but also to invite its occurrence. Acute destructive myelitis has a tendency to displace or distort the gray matter, and this condition may be made still further grotesque by handling the cord at the autopsy.

Chronic myelitis, compression myelitis, poliomyelitis anterior, tumors and cavities, may all induce distortions or asymmetrical conditions of the substances of the cord, which may be enhanced by careless manipulation at the autopsy, and should not be confounded with congenital malformations.

2. *Asymmetries of the cord with congenital defects of the extremities or muscles.*

These acquired asymmetries are not clearly enough separated from the congenital malformations of the cord. There seems indeed to be a tendency to look at them as arising from defective development of groups of ganglion cells, whereas the asymmetry arises from the gradual post-natal atrophy and lack of further development of the group of ganglion cells corresponding to the defective member. They should be regarded as due to an arrest of development at birth rather than to a failure of development.

In cases of intra-uterine amputations which have lived a number of years, the corresponding portion of the cord is asymmetrical. The gray and white matter, particularly the former, is shrunken on the corresponding half of the cord.

What occurs in the motor part of the cord in a case of intra-uterine amputation seems to be somewhat as follows: The ganglion groups of the absent limb develop equally with the surrounding groups up to near the time of birth; they send out their neural processes in company with the other cells which become nerves destined for the absent member. After birth their development is retarded from disuse, and in the course of years they disappear and cause a shrinkage of the gray horn. In like way the nerve fibers atrophy in the nerve roots.

Examination of such cases have been made by Edinger (2) and Davida (3).

A similar but much more slightly marked asymmetry of

the cord is found a number of years after *ordinary surgical amputations.*

Cases of *congenital club-foot* have been described with slight asymmetry in the adult cord, due to a trifling shrinkage of the anterior horn on the corresponding side. Although any discussion as to the peripheral or central origin of club-foot is foreign to this paper, the writer believes in the former view, and that the resulting secondary changes in the cord take place in the same way as in intra-uterine amputations, only that they are of a corresponding lighter degree of intensity. The condition of the spinal cord in club-foot is described by Ross (4), Marchand (5), Dejerine (6), and Cogne and Troisier (7).

Congenital absences of voluntary muscles, or other defective developments of the extremities, would produce similar slight asymmetries in the adult cord.

An asymmetry of the gray matter above or below a tumor of the cord may result from the enlargement of one of the horns due to an additional amount of gray matter forced upward or downward by pressure from the tumor. A good example of this is reported by Adamkiewicz (8).

Perhaps a congenital defect in the cord itself has been too sharply excluded in these asymmetries associated with club-foot, intra-uterine amputations, and the like, but I am disposed to think in these cases that the cord is very nearly normal near the time of birth, and that the cord changes take place in later years of life by atrophy of ganglion groups from arrest of their further development.

Serres (9) found in two embryos without lower extremities an absence of the lumbar enlargement, an observation which would tend to militate against the view expressed above; but as he examined the embryos at a period of development before the enlargements appear, the observation has not much value.

Tiedemann (10) examined a case of congenital absence of all four extremities a few days after birth, and found the enlargements of the cord very defective and that the cord had but half its usual volume in these regions. The case was not examined microscopically and the condition of the gray matter is not stated. As many of the fibers conveying centripetal impressions to the cord develop in the periphery and

grow inward to the cord, the cutting off of the large supply from all four limbs ought to contribute materially to the reduction of the volume of the enlargements. Troisier (11) describes a case in which a congenital absence of the left hand and neighboring part of the forearm was associated with an asymmetrical condition of the cord in the lowermost cervical region in a child dying six months after delivery at the end of the eighth month. The author excluded intra-uterine amputation, and believed that the absent member was due to a simple arrest of development. The changes in the cord (shrinkage of the anterior horn and white matter on the left side in the lower cervical region) seem entirely secondary and indicate atrophy consecutive to the absent member. In Pick's (12) masterful article on Agenesis of the Spinal Cord a case is recorded in which the spinal cord of a pig, having one congenitally defective extremity, examined six months after birth showed an asymmetrical condition of both the gray and white matters, in the corresponding enlargement of the cord, similar to Troisier's case (Troisier's case is discussed by Pick (12) and Leyden (13)).

3. *Variations in the Volume of the Cord as a Whole.*— Variations as to the length, thickness, position in the vertebral column, and configuration of the external furrows and fissures occur, apparently dependent upon the variations in the physical development of different individuals.

Observations of the measurements of the segments and intersegments, the number of entrance funiculi of the nerve roots and their anastomoses, show variations which will be described in a future paper. Any one who has examined the human cord extensively will have noticed these volumetric variations, but we have not yet observations enough to explain them very well.

A review of a number of personal autopsies would tend to show that the cord is liable to become flattened, with prominence of the external furrows, especially in the dorsal region, in old persons.

After this cursory consideration of spinal-cord malformations in general, a paragraph will be devoted to the frequency of heterotopia before going on with the details of the subject.

As a matter of fact, true heterotopia of the spinal cord, as already stated, is exceedingly rare. Pick, in 1878 and 1881, has published four instances, the only genuine cases of heterotopia of the gray matter, and two cases of heterotopia of the white matter in the medulla, or abnormal nerve-fiber bundles, have been described by Pick and by Cramer in 1890. These six cases are the only true examples I can find of heterotopia of the cord or medulla in the literature. Besides these, two personal cases of true congenital heterotopia—one of the gray matter and one of the white—will be described later. The other cases in the literature of the subject are not true cases of heterotopia.

Even on *a priori* grounds, without any analysis of the cases or experimental work, we should not expect any such bizarre distortions as are described in some twenty-five cases to exist preformed in a normally formed individual without some index of their presence. Localization is too rigid, and the two substances of the cord are altogether too delicately and constantly arranged with reference to each other, for us to believe that any considerable distortions of the gray and white matter may exist without some deformity elsewhere, or some other clew to the cord changes.

On the other hand, it is plausible enough that small bits of the gelatinous substance of Rolando might be here and there displaced in the developing cord, and persist in the adult without recognition, or that a tiny lump of the more highly organized gray matter containing ganglion cells might be partially separated from the parent horn by a few intervening vertical nerve fibers of the white columns, and yet go on performing its functions. But should the grosser malpositions of the gray matter, or the larger portions of extra gray matter, or the partial doubling of the cord, occur in its development, these malposed or extra portions of the cord would dwindle away in later life from functional inactivity, unless associated with some corresponding peripheral condition. The little clusters of ganglion cells found in a section straying out into the white matter on the confines of the gray matter in animals, such as oxen and horses, and occasionally in the human cord, can hardly be called heterotopia.

We wish to show eventually that, excepting the cases of Pick, Virchow, and Cramer, the great majority of the cases are simply dispersions of the substances of the cord by bruising done in its removal at the autopsy.

Section II.

AN ANALYSIS OF THE PUBLISHED CASES OF TRUE HETEROTOPIA OF THE SPINAL CORD AND MEDULLA OBLONGATA.

I. The true cases of heterotopia of the gray matter.

II. The true cases of heterotopia of the white matter.

Analysis of the Published Cases of Heterotopia of the Spinal Cord.

Of the thirty-one cases of malformations or of heterotopia of the spinal cord, the only genuine cases are the following six cases. Four of them are malformations of the gray matter of the spinal cord, and two are malformations of the white columns in the medulla oblongata. To each of these is added a personal case. Malformations associated with monstrosities are of course excluded from this group of cases.

The case of Virchow is more properly an instance of asymmetry rather than heterotopia, but it is most conveniently instanced in this section.

1. *The True Cases of Heterotopia of the Gray Matter of the Spinal Cord.*

Pick's* (5) *Cases, No. 1, 1878.—The cord was taken from a well-formed man dying with progressive muscular atrophy. In the course of the microscopic examination of the cord a minute lump of gray matter only one millimetre in diameter was found misplaced in the posterior white columns of the lumbar region. (See Fig. 1, tracing of the original plate.) The mass was so small that only a few sections could be cut of it, and, as the sections passed through it, the misplaced lump became smaller, and finally ended in the neuroglia strands of the posterior columns. This was the only heterotopic mass in the whole cord. It contained nuclei, and had the structure of the gelatinous substance of Rolando.

This tiny heterotopic mass was sharply outlined and sent out one or two branching processes of its substance which became continuous with the neighboring glia strands. Medullated nerve fibers passed through it (apparently the

FIG. 1.—Pick's case of heterotopia of the spinal cord. The shaded circular area in the posterior column indicates the heterotopic fragment of gray matter.

fibers of the internal division of the posterior roots, as the mass stood directly in their path). Finely medullated nerves also passed through the mass vertically. The author, knowing that the mass might have been produced by pathological processes, carefully excludes sclerosis, tubercle, and any form of tumor.

This case of Pick is a genuine instance of heterotopia. The white matter of the cord at the level of the heterotopic mass is perfectly normal; it shows none of the appearances of crushing or bruising, which will be seen later almost always accompanying artificial displacements of the gray matter. There is but this single isolated fragment of gelatinous substance misplaced during the development of the cord.

Second Case of Pick, 1881 (15).—The second case of Pick relates to a fifteen-year-old boy having but the intelligence of a child. There was nothing notable about the motility or sensibility of the extremities of this idiot, or about their development, except that he was left-handed. There was considerable scoliosis of the vertebral column, and the brain was hemi-atrophic, while the cord seemed normal grossly. The latter was examined microscopically, however, and at the junction of the dorsal and lumbar cords a minute misplaced mass of gelatinous substance of Rolando, containing ganglion cells, was found in the right posterior white column. In places the misplaced mass joined the posterior horn by extensions or processes of gray matter. The mass was so small that only twenty sections could be cut through it.

It is unfortunate that the description of the other structures of the cord at the level of the misplaced mass, especially the surrounding white fibers, is not more elaborate and accompanied by drawings so that bruising of the cord could be positively eliminated; but I gather from the description that the appearances were very much like those in the first case (see Fig. 1), and that the case is an authentic one of heterotopia of the gray matter.

Third Case of Pick, 1881 (15).—The third case was taken from a woman aged twenty-nine, who had attacks of unconsciousness lasting half an hour with tremors in the arms. The patient's hands and feet became paralyzed, which persisted until death.

Microscopical examination of the spinal cord showed the presence of vacuoles in the ganglion cells—explaining the paralyses. There was no myelitis or other lesion of the cord. Misplaced fragments of gray matter—gelatinous substance—were found in both the lumbar and cervical regions. In the lumbar cord there were two symmetrically (?) misplaced bits of gelatinous substance at the same level, in the front part of the posterior white columns on either side of the posterior septum. One of the displaced masses was nearer the posterior horn than the other, and was attached to the same by narrower and broader bands of gelatinous substance. For the most part this latter mass had a rounded form, but in some sections it was more or less stretched

out. Altogether, these two little masses were just like the displaced mass in the preceding case in character and position, except in the absence of ganglion cells.

Two other masses of the same structure and also nearly symmetrically situated at the same level were found in the posterior white columns of the cervical region, about where the internal division (?) of the posterior root fibers enter the posterior horn.

This third case of Pick is also one of true heterotopia, but it would be still more acceptable if drawings were present and if the description was not so limited to the misplaced masses, and was more complete about the condition of the other parts of the cord in the same region, which is so necessary in discriminating between true heterotopia and artificial distentions of the gray matter. A glance at Figs. 14 and 15 will explain just what is meant in this connection. Both of these plates show mere bruises of the cord, yet if the description were limited to the displaced mass entirely and omitted the condition of the surrounding tissues, the impression would be strongly conveyed that the case was one of true heterotopia.

Fourth Case of Pick, 1881 (15).—This case came from a girl, seventeen years old, with paraplegia from compression of the cord from osteitis of the thoracic vertebræ. There were also spinal pachymeningitis, tuberculosis of the brain, and myelitis of the middle dorsal region. A heterotopic fragment of gray matter was found of the same description as in the second case.

Virchow's Case, 1887.—Hans Virchow (9) describes an asymmetrical condition of the cord in a case of congenital hydrocephalus internus with microcephalia. In the fifth dorsal segment the right posterior horn was displaced laterally so that the exit of the horn at the periphery of the cord was situated about twice as far outward from the sæptum posterius as in the case of the left posterior horn exit, which was normally situated.

This asymmetry could have been produced artificially so easily (see notes on the hardening and removal of the cord in Chapter III) that I am not at all positively convinced that the displacement was a preformed condition of congenital origin. It would seem so, however, for the author does not

note any of the other changes in the cord tissues indicative of bruising or faulty manipulation, nor are there any appearances in the plate illustrating the displaced horn characteristic of an artificial origin.

The Writer's Case.—The condition was discovered accidentally in the course of an examination of the nervous system in a case of myxœdema. The heterotopic fragment —but half a millimetre in diameter—was composed of gelatinous substance containing a few nuclei, but no ganglion cells or nerve fibers. *It was situated just posterior and external to the lateral angle of the nucleus cuneus.*

The surrounding white and gray substances were everywhere normal, and there were no evidences of bruising. The displaced mass looked more like an extra portion of gelatinous substance than a detached portion of the nucleus. This was the only instance of displacement in the cord or medulla, which were perfectly normal and exhaustively examined. The case is very briefly alluded to by Hun and Prudden, Myxœdema, *American Journal of the Medical Sciences*, July and August, 1888. This is the only instance of the condition ever seen by the writer among one hundred or more cords examined microscopically.

2. *The True Cases of Heterotopia of the White Matter in the Medulla Oblongata.*

I can find no instances of congenitally misplaced white matter in the spinal cord, but two cases of abnormal bundles in the medulla oblongata are reported, which will be supplemented by a personal case of the same.

Pick's Case, 1890 (17) (*illustrated*).—In a hundred medullas examined by Pick, one was found containing a sharply defined abnormal bundle of nerve fibers. The bundle appeared on one side only, and, taking its origin in the remains of the lateral column at the level of the upper part of the motor decussation, it passed inward and in front of the gelatinous substance of the posterior horn, and finally joined the corpus restiforme in the upper portion of the medulla without connecting with any nucleus or set of fibers on its way up.

Pick is careful not to confuse the abnormal bundle with the solitary fasciculus, and very aptly considers the mis-

placed bundle as part of the connection between some of the lateral white column fibers and the corpus restiforme. The writer cites Henle's allusion to a very similar bundle in his *Handbook of Anatomy*. Henle was evidently not perfectly clear about the abnormal bundle, for in the second edition of the work he calls it the respiratory fasciculus.

Cramer's Case, 1890 (18) (*illustrated*).—Cramer accidentally found in the course of an examination of a medulla from a case of cerebellar hemiatrophy a compact misplaced bundle of nerve fibers very much the counterpart of Pick's abnormal bundle. The abnormal bundle seemed to originate in the remains of the lateral column of the cord, and, as it passed inward and forward of the Rolandic substance of the posterior horn, it gathered in some of the skeins of fibers of the lateral column remains.

The bundle reached its greatest development at the sensory decussation, and in the midolivary region it was situated just ventrad of the solitary fasciculus, in the path of the recurrent vagus strands, in which position the bundle was split into two component columns. In passing up higher in the medulla, the bundle gradually disappeared by dividing up into fine bundles which tended to pass toward the corpus restiforme. The solitary fascicles were well formed in this case, and the writer thinks the bundle conveyed fibers from the lateral column to the corpus restiforme, which always derives some of its fibers from that column.*

The Writer's Case.—This case may be very briefly described. The abnormity was found in the medulla of an

* A third case of abnormal bundles of white fibers in the medulla, described by Kronthal (19), unfortunately escaped the writer's notice until the text reached the printer. In making serial sections of a medulla of a case of bulbar paralysis Kronthal found two abnormal circular bundles of fibers of limited vertical extent lying mesal of the hypoglossal nucleus, near the floor of the fourth ventricle. One of the bundles measured 240 μ and the other 160 μ. After thirteen sections had been cut the two bundles fused into one, which could not be traced any farther. (In examining a medulla of a case reported by Dr. Delavan—Further Investigations as to the Existence of a Cortical Motor Center for the Human Larynx, *New York Medical Journal*, June 22, 1889—the writer found two unusual bundles of fibers in the inner margins of the hypoglossal nuclei, which were regarded merely as some stray bundles of the posterior longitudinal fasciculus, or bundles of association fibers for the cells of the hypoglossal nuclei.)

old tabetic, and consisted in a malposition of the left respiratory fasciculus. The upper portion of the fasciculus deviated from its usual position by being placed lateral and slightly ventral. It was quite close to the corpus restiforme. Besides this, the fasciculus, maintaining misplaced lateral position, as it passed downward could be traced to a much lower level as a compact bundle than its fellow.

Section III.

A CONSIDERATION OF THE METHODS OF REMOVING THE CORD AT AUTOPSIES, AND THEIR RELATION TO THE PRODUCTION OF ARTIFICIAL MALFORMATIONS BY BRUISES.

It is not easy to remove the adult human spinal cord by any of the practiced methods without using a considerable amount of force, which is difficult to control perfectly so as not to injure the cord itself. The spinal cord is quite universally removed by the median dorsal incision over the spinous processes, and, after exposure of the laminæ, these are sawed through with either a single-bladed or a double-bladed saw. After this a hook is inserted under the laminæ, or a spinous process is seized with a strong forceps, such as the lion-toothed kind, and the connected posterior archway of the vertebral column torn off with a sudden jerk.

If all the laminæ have been sawed through completely, the cord lying in the dural sac is exposed without much liability of injury. The only danger of wounding the cord in this procedure is that the saw may slip through the laminæ with a sudden thrust and strike the tough dural sac, distributing the pressure to the cord without the operator's realizing it. If the saw does pass through into the vertebral canal, the operator can not tell whether it may wound the cord or not. The canal is spacious enough, so that the saw-point may enter and not touch the dura, or it may slip down alongside of the dura without doing any harm, or it may cut into the dura and cord, or bruise the latter more or less.

This paragraph may seem trivial, and it is true that the chances of wounding the cord, especially one of normal consistence, by the saw alone, in the hands of a fairly skillful operator, are very slight. But, in a cord of diminished

consistence, a slight tap on the dura mater from the saw may produce extensive changes in the distribution of the softened cord substances, often without any external alterations, and the effects of the bruise will not be discovered until the cord is examined microscopically.

To avoid bruising the cord, it is so important to use the saw almost entirely in dividing the vertebral arches that a properly shaped instrument should be used. A slightly curved saw, or one gently rounded at the point, operates much better than the rather frequently used perfectly straight saw, which is awkward in the lumbar and cervical regions, because the point becomes impacted in these curved regions of the vertebral column. The double saw with adjustable blades has no especial advantages over the single-bladed instrument; the double saw, it is true, operates more rapidly over the prominent dorsal region, but it is liable to become impacted in the bony saw grooves in other regions, so that in dislodging it the operator has to use sudden, jerky, forcible thrusts, which may drive the blades through into the vertebral canal and injure the cord.

With the slightly curved saw the operator is less dependent upon the subsequent undesirable procedures with the chisel and osteotome than with the perfectly straight blade.

Thus, if the posterior vertebral archway is completely sawed through without using the chisel, the danger of bruising the cord is reduced to a minimum. The best way is to test the spinous processes one by one manually after finishing with the saw, and if they are all freely movable the loosened posterior archway may be torn away with the hook or forceps.

If any of the spinous processes are immobile, it is best to use the saw again rather than the chisel and mallet.

As a matter of fact, in practical autopsy work the laminæ are not often completely sawed through, as just described. In an emaciated subject, where the spinal column is superficial and readily reached, it is a comparatively easy thing to separate completely the vertebral archway with the saw alone; but in the more deeply placed vertebral column of fat or muscular subjects, especially in the lumbar region, more difficulty is encountered, and one or more of the ver-

tebral arches are left incompletely divided, inviting the use of the chisel.

So in practical work some portion of the arches is generally incompletely separated from the vertebral bodies by the saw, and it is then customary to use the chisel and mallet; and this is the proceeding that bruises the cord and gives rise to the dispersions of the cord substances so universally mistaken for congenital malformations, or the results of pathological processes.

After breaking through the incompletely sawed laminæ by blows from the mallet on the chisel fitted in the saw groove, the operator tears away as much as possible of the vertebral archway, and if some of the arches still remain unseparated, which is very likely to be the case, he may repeat the chisel-and-mallet procedure or use large or powerful bone-cutting shears.

These shears are of various kinds, but all of them have such thick jaws that their mere introduction into the vertebral canal may be compromising to the integrity of the cord, while the subsequent wrenching and twisting of the bones in the process of cutting menaces the cord still more seriously.

The chisel or other instruments may also be used as levers to pry up remaining laminæ, in which case the cord is liable to be bruised by the slipping of the instrument, or the sudden depression of the bone used as a fulcrum.

The most extensive bruises are, in my experience, made by the use of the chisel. Even if the chisel has shoulders to prevent it from passing directly into the spinal canal against the cord, it is liable to fracture the arches and drive down fragments of bone against the dura. The suddenness of the blow from the mallet or the hammer also conduces much to the intensity of the dispersion of the cord substances. We shall see later that quick, sharp blows tend rather to produce more grotesque alterations in the cord matters than more deliberate pressures. The intervention of the dura-matral covering between the force of the blow and the cord also modifies the effects of the blow. The adult human dura mater is so thick and tough that it is a very important factor in the production of the cord bruises. It is hardly ever cut through by the autopsy instruments,

and thus the cord is bruised instead of being lacerated or incised.

In the smaller animals—such as cats, dogs, monkeys, etc.—the dura mater is much thinner and more delicate and seems to fit tighter about the cord, so that in these animals blows from sharp autopsy instruments tend rather to produce simple lacerations than bruises with complex dispersions of the cord substances. The more fragile construction of the vertebral column also requires less force in removing the cord. At the same time, moderately complex spinal bruise artefacts occur occasionally by the operation of the scissors or osteotomes, which are most convenient in these animals, but are hampered in their action by the small lumen of the spinal canal. The conditions relative of bruises of the cord in the smaller animals are quite similar to the cases of children or infants at term. The bony canal and the dura mater here also play a less important part in the production of bruise artefacts than in the adult. Blows in the autopsy procedures, in such cases, give results more like those struck with cutting instruments on the entirely naked cord described in the next section. Furthermore, the tunics of the cord itself, in very young persons or small animals, have such a delicate structure that they are not favorable for distributing sudden concentrated force so as to produce the more complex dispersions of the two cord substances.

Thus it may be said, in general, in removing the spinal cord in such cases, that it is more frequently simply cut or torn rather than bruised, and if the cord does become bruised from the deliberate pressure or pushing by the scissors or osteotome, the results are usually less complex than in adult cases.

On the other hand, in larger animals like horses or oxen, the cord is even more difficult to remove and the tendency toward complex bruise artefacts increases. The vertebral canal is stronger, the instruments have to be used more forcibly, and the thicker and tougher coverings of the cord also contribute their share in the production of the mechanical artefacts.

These detailed remarks about the occurrence of bruises of the cord in animals seem not inappropriate, for such

spinal-cord bruises in animals have been described in the literature as congenital malformations without any reservation at all as to the artificial origin of the changes.

Besides this ordinarily practiced method of removing the cord, there is another way of removing it by peculiar cutting chisels devised by Brunetti, which do away with the time and trouble of using a saw. By this method, which, I believe, is practiced considerably in Vienna, and is used more or less in other places on the Continent, the cord is taken out through the abdominal opening by the removal of the vertebral bodies. The method is said to be very expeditious, and has the further advantage, valuable in certain limited or private autopsies, of doing away with the disfiguring dorsal incision of the other method.

The chisels for this method are about fifteen centimetres long and a centimetre and a half wide; the cutting end is flat, like an ordinary carpenter's chisel, while the opposite end is heavy and cylindrical. The cutting edge is V-shaped; one shorter arm of the V is a pointed rounded guard or shoulder, while the other longer arm is the beveled cutting edge of the chisel. There are two of these chisels—a right-handed and a left-handed one—and they are used in this fashion: The pointed guard is inserted in the vertebral canal between the pedicles on one side, with the cutting arm resting against the pedicles, so that the long axis of the chisel is parallel to the vertebral column. Then the pedicles are chopped through one by one on either side, according as the right or left chisel is used, by blows from the mallet. The chisel is directed so that the force of the blow is not directed inward toward the vertebral canal, but parallel to it.

These same chisels are also used in conjunction with the saw to sever the laminæ when the cord is taken out by the dorsal method.

I am unable to say much about the chances of damaging or bruising the cord by this method, never having used this new ventral method or the chisels which it requires, but I should think that the use of these chisels, either ventrally or dorsally, would be very liable to damage the cord and to bruise it very thoroughly.

To find out the effects of the use of these chisels on the

cord, one of my colleagues, familiar with these new methods, removed a cord for me by these chisels. This cord did not show many signs of bruising in the fresh external appearances, but in the subsequent examination a good part of the cervical enlargement had been considerably bruised with the resulting bizarre dispersions and malpositions of the cord substances. It must be said that the operator, an expert pathologist, acted in the best of faith in removing the cord carefully, for the case was an interesting one of hydrophobia, requiring a medico-legal autopsy and microscopical examination.

In addition to the instruments already mentioned, one or two other rather obsolete weapons are supposed to be of service in removing the cord rapidly, by enabling the operator to dispense more or less completely with the saw. These are the chisels of Esquirol and Amussat. Esquirol's chisel is a doubled-bladed affair, with convex cutting edges, which are adjustable or composed of a single piece of steel. The chisel of Amussat is curved; the blade is set at an angel of 45° with the handle and the blow is struck in front of the blade, so that the instrument is a sort of hook and chisel combined. Neither of these antiquated tools, especially the former, should be used unless one wishes to study the effects of bruising the spinal cord.

Independent of the autopsy procedure, the topography of the cord substances may be disturbed by careless hardening or manipulation in dissecting the cord. In a late autopsy in very warm weather, when the consistence of the cord is diminished so that it is soft and pulpy, the mere handling of the cord after the autopsy preparatory to putting it in the hardening fluids has a tendency to interfere with the topographical relations of the cord substances. Pinching such a cord with the fingers, and feeling of it repeatedly to test its consistence, disturb more or less the arrangement of the cord tissues.

In separating the attachments of the dura mater and cord after the vertebral canal is opened, great care should be taken not to touch the cord with the hands at all. The cord should not be bent, twisted, or stretched too severely. Every time a cord of diminished consistence is touched or squeezed or tested with the fingers, a certain amount of

damage is done, which, although not sufficient to produce the changes simulating malformations, may produce minute changes or modify pathological alterations so as to interfere very seriously with proper interpretation at the microscopical examination.

Some pathologists open the dura mater with scissors (which sometimes wound or cut the cord) and remove the cord, leaving the dura mater in the spinal canal. It is safer, however, to take the cord out with the dura and to use instruments almost entirely in the operation, such as small scissors, forceps, and scalpel. In this way any undue accidental force or traction is distributed over the whole length of the spinal cord by means of the dura, and the resulting damage to the cord reduced to a minimum. Of course, the cord is also liable to be bruised or show minute artefacts if carried about from the autopsy mixed up with the other fresh organs in a common receptacle.

When a soft cord is cut into segments transversely, the gray and white matters tend to well up out of the cut ends, especially the white matter. The latter will flow out at the periphery of the cut surface and turn over the pia-matral covering. In such a soft cord, when divided transversely, the slightest manual pressure thrusts out still more of the cord substances, and if the dura mater is attached they are also pushed out by the traction of the ligamentum dentatum, when the dura is pulled, folded, or twisted. These disarrangements of the cord matters, in places where the cord has welled out of the pia mater when cut or divided transversely, are quite frequent and fairly constant in their appearance; and, in passing, Figs. 5, 6, 8, and 9 may be referred to as showing the changes of this kind.

These remarks about the effect of handling the cord are still more applicable to cords which are the seat of myelitis or other lesions diminishing the consistence of the cord. In acute destructive myelitis the slightest touch, or the operation of dividing up the cord, pushes the cord matters about, especially the gray, into wrong places.

The treatment of the cord while hardening, especially if it is soft or pulpy, may tend to produce misplacements, and still more frequently asymmetries, of the cord matters.

If the cord has a twisted position or is carelessly doubled up in the hardening fluid, or even if it presses by its own weight against the bottom of the hardening jar, there is also liability of producing these artificial displacements or asymmetries. The welling up of the tissues from cut surfaces of softened cords may be increased during the early stages of the hardening. The dura mater should not be hardened with the cord, as it shrinks or stiffens up a little in the hardening, and squeezes the cord a trifle by transmitting pressure to the filaments of the ligamentum denticulatum.

The best way to harden the cord perfectly is to remove the dura mater and make many transverse incisions, and then curl it up in a loose spiral, so that the cuts gape open, and lay it out flat on a wad of absorbent cotton in Müller's fluid. When very perfect symmetry of the gray horns is wanted, as for measurements, or in a case after amputation, I have found it best to hang the cord up vertically without the dura in long glass tubes, with just weight enough on the cauda equina to keep the cord straight. Very soft cords are treated in the same way.

Cords softened by myelitis are hung up in this way and are not incised at all in the damaged regions, but various proportions of alcohol, from five to fifteen parts, are added to a five-per-cent. solution of bichromate, which gives it a greater penetrating power and a more rapid action, to compensate for the absence of transverse incisions. After a week or two, ordinary bichromate solutions may be used. If softened myelitis cords are cut open the cord flows out, and they can not be transported from the autopsy room without damage, and it is impossible to keep the cord matters topographically intact in the hardening. But if hardened *en masse*, as just described, artificial malformations are obviated, although the preservation of structural details and the staining of the sections may be slightly imperfect. It is bad practice to harden the cord in the same jar with portions of other organs which may press on it.

General Remarks on the Production of Artificial Malformations of the Cord by Technical Methods.

As a matter of fact, it is very easy to bruise the spinal cord at the autopsy, and it is quite frequently done. In

examining a laboratory collection of about seventy-five human spinal cords taken out for suspected lesions in ordinary routine autopsies, seven of these showed effects of bruising. On the other hand, a great deal of force and hammering with the chisel and the mallet (in opening the spinal canal) may not disturb the cord at all. It is all a matter of accident. If the chisel is used, the liability of bruising is greater, and the operator may not be aware of having damaged the cord. The dura is generally intact after a bruise, and does not indicate it. The cord may show no gross, or but trifling external changes, or it may be very much altered, as hereafter described.

At the autopsy the chisel and the shears do the greatest and almost exclusive damage to the cord, and the ordinary chisel used in the dorsal method of removal does the most harm, for the force of the blow is directly toward the cord. The most skillful operator (if he uses the chisel or osteotome) is liable to bruise the cord at any moment, and produce the attendant appearances suggesting congenital malformations, or the results of pathological processes.

The writer, even after much experience as to how these bruises occur, in hastening the cord removal with chisel or osteotome, has accidentally damaged the cord so as to interfere with the understanding of the lesions in the microscopical examination of valuable material.

Brunetti's chisels for the ventral removal of the cord are open to less objection than the ordinary one, for the direction of the blow is not against the cord, but alongside of it. However, it must require a very practiced operator to use this method without contusing the cord.

Even if not mistaken for congenital malformations, these bruises of the fresh or hardening cord interfere greatly with the appreciation of pathological processes, if they happen to be present in the contused part, during the microscopical examination. Both the distribution and structure of the lesions may be so much altered that the bruised part has to be discarded.

It will also be shown later that bruises of the normal cord may produce appearances simulating sclerotic patches or other spinal lesions, and, inasmuch as the whole subject is misunderstood, these have very likely been described or

considered as due to pathological processes instead of the bruises.

When the bruised cord is examined microscopically, some time after the autopsy, by a different observer knowing nothing about the autopsy accidents, the disposition is natural enough, at first glance, to regard the changes as preformed malformations, and it is fair to suppose that some of the published cases have come about in this way.

After all that has been said, however, about the production of these bruises by technical methods, when looked at practically, although the minor contusions occur with tolerable frequency (once in seven to ten cases), the grosser distortions are very rarely produced. Instances of perfect doubling of the cord have been recorded but twice in the literature. These were most undoubtedly caused artificially. Partial cord duplication, caused by bruises, is described but two or three times.

The text-books on autopsy technique do not explain at all the extensive and deceptive artefacts due to cord bruises, and do not warn the reader forcibly enough about the operations which are liable to produce bruises of the cord. Orth (20) makes some very valuable general remarks about advising the operator to be very cautious about injuring the cord in any stage of its removal, but fails to enforce his suggestions by explaining the serious and complex results of these injuries, whether slight or severe.

Nauwerck (21), on the other hand, in a most recent work on the methods of performing autopsies, tends to ignore the danger of wounding the cord, and advocates the use of the chisel freely. He considers the instrument indispensable, and on page 10 indicates that it saves time to dispense with the saw altogether by breaking through the vertebral arches from beginning to end with an ordinary carpenter's chisel.

This is very bad advice indeed. The less the chisel or osteotome is used the better. If these instruments must be used, the most skillful operator should remember that, at some time or another, he is quite certain to bruise the cord, and produce misleading changes in both the topography of the gray and white matter, and in their minute structures.

This latter set of changes sometimes ruins the cord for subsequent examination, for if these artefacts, according to the present tendency, are not entirely mistaken for the features of pathological processes, it becomes exceedingly difficult to distinguish artefacts from lesions when both are mixed up together in the same section under the microscope.

In removing the cords of infants, new-born children, or small animals with fragile or partly cartilaginous vertebral arches, skillfully used slender-bladed scissors or osteotomes are better than the ordinary saw.

In the fœtus, the whole technique of removing and hardening the very easily damaged cord requires a still more delicate and cautious treatment. It is often better in such cases not to attempt to remove the cord at all, but to harden it *in situ* by cutting out the vertebral column entire. Then small openings should be made here and there in the arches, or the cord with the vertebral column should be cut into segments, to permit the hardening media to penetrate thoroughly.

Section IV.

DESCRIPTION OF THE APPEARANCES OF NON-INTENTIONAL BRUISES OF THE CORD, SELECTED FROM ORDINARY ROUTINE AUTOPSIES.

These cases have been found in examining a laboratory collection of spinal cords comprising about seventy-five cords removed in the last fifteen years for suspected lesions, and about twenty-five or thirty normal cords. These have been removed by the dorsal method, and, as a whole, the collection has been very skillfully removed, as shown by the small percentage (about ten per cent.) of contused instances found.

It seems wise, before dealing with the experimental bruises of the cord, to describe these cases, because just such instances as these, bruised entirely unawares at the autopsy, must form a certain proportion of cases in any extensive laboratory collection of spinal cords, and they seem to be found accidentally from time to time in the course of microscopical examination, and are described as malformations.

Case I. Figs. 1, 2, 3, 4, 5, 6, Plate I.—This case first enlisted my attention to the subject of bruise artefacts, and the cord was sent to the writer for examination by Professor Hun, of Albany, in 1887. The clinical history does not relate in any way to the changes in the cord which have been produced by accidental bruising, but an abstract is given, so that the reader may not have to take this assertion for granted.

The cord was taken from a man, aged thirty-eight, who had no bodily deformities. "Symptoms began in 1882 by tingling, heaviness, and awkwardness in the right leg. In 1883, same symptoms in the right arm. Symptoms grew worse, and in 1884 incapacitated him for work. The peculiarity of the symptoms was that he could perform automatic movements well, but voluntary movements only very imperfectly. In walking, the right leg seems rigid and drops a little, but he can run well and swiftly and can jump over a chair after a fashion. The patient explains this by saying that in running he gets his mind off the leg, but when he gives his attention to the leg and tries to walk, walking becomes very difficult.

"When asked to flex his left arm at the elbow, the biceps contracts firmly; when he tries to do the same with the right arm, the biceps contracts feebly at the first attempt, and at each subsequent attempt contracts more feebly and the arm is thrown into a tremor, until after three or four attempts it does not contract at all. When he tries to put his hand on the top of his head, the first attempt succeeds well, but on the second and third attempt the hand trembles very violently and the movement becomes weak, and finally impossible. When resistance is offered to any of these movements, even when he can no longer make them without such resistance, the contraction becomes very strong and the patient's strength increases with the resistance offered.

"In the same way, when the patient grasps the experimenter's hand with his right hand, the grasp is weak when no resistance is offered, but when the experimenter returns the grasp strongly, then the patient's grasp becomes so strong as to cause much pain—indeed, unbearable pain—in the experimenter's hand. It seemed as if the resistance called forth strength in a reflex way, which the volition of the patient could not produce.

"Sensibility of arm and leg normal. Much twitching of muscles of body, especially on right side.

"These symptoms were present in the early part of December, 1886; in the latter part of December he began to fail and lose flesh rapidly, and died January 3, 1887."

The *autopsy* was made sixteen hours after death and contains these notes about the brain and cord:

Cerebral dura and pia mater normal. An unusually large vein containing a white coagulation, apparently of post-mortem origin, ran up in the fissure of Rolando on the left side. Brain otherwise normal. Spinal cord seems softer than normal, *especially in lower part, and the gray matter in certain sections seems unequal* on the two sides, sometimes in the posterior, sometimes in the anterior horn. Especially in the dorsal region the right anterior horn seems smaller than the left."

There was nothing abnormal about the other organs and no malformations were found anywhere in the body.

I did not see this cord until after the hardening was completed, and can not describe the fresh gross appearances, and there are no notes in the autopsy record about any accidental bruising of the cord, but the hardened specimen showed a contusion in the dorso-lumbar junction. Here the cord was much enlarged, the enlargement being fusiform, and in places the white matter has been thrust out through splits in the pia mater and nodular and diffuse portions of it appeared uncovered on the surface of the cord. This contusion involved the twelfth dorsal and first lumbar segments. The transverse enlargement in the bruised place was about one and a half times the normal diameter of the cord in this region. I afterward learned from Professor Hun that the bone-shears had been used freely in getting out the lower portion of the cord, which explains the occurrence of the dorso-lumbar contusion.

Above and below the bruise the cord is not much smaller than usual in its gross appearances, whereas generally the enlargement of the cord from a bruise is at the expense of the contiguous portions.

The asymmetries of the gray matter described in the autopsy notes are also indicative of blows more diffuse and less violent in their effects than in the large circumscribed bruise in the dorso-lumbar region. In the hardened cord there were also places where the cord substances, particularly the white matter, had flowed out from or had been thrust up by pressure out of the transverse incisions. Beyond the features of the lumbar bruise, the external gross appearances were perfectly normal.

Microscopical Examination of the Dorso-lumbar Bruise.—Fig. 1, Plate I, shows the appearance of a section of the cord in the uppermost level of the bruise. A portion of the white matter has been thrust up from below by the force of the blow and splits open the right posterior horn. The fibers of this displaced white matter in the posterior horn do not run vertically, but are crushed up together in a confused mass, with the

majority of the fibers running for a short distance parallel to the plane of the section. The axis cylinders of this displaced white matter are distinct, but the myeline of the fibers is badly disintegrated, so that the area stains a trifle lighter than the surrounding normal white matter.

Fig. 1, Plate I, should be compared with Fig. 24 (Plate VII), in which the cord was bruised intentionally with the production of results quite similar to those shown in Fig. 1. Fig. 24 shows macroscopically the actual transit of a mechanically displaced column of white matter, and how it splits open the anterior and posterior horn which are shown in section in Fig. 1.

Another portion, apparently, of this displaced column of white matter radiates out from the outer segment of the split horn to the periphery of the cord, the fibers running horizontally. A few of these latter fibers in other sections break through the superficial neuroglia and pass outside of the cord, and correspond to the extraneous portions of white matter noted in the gross appearances of the bruised region.

A few millimetres below the level the bruise has dispersed the cord substances as shown in Fig. 2, Plate I. Here the horns on both sides are split open by displaced columns of white matter driven from other levels into the gray matter. The right posterior horn resembles its arrangement in Fig. 1, but in Fig. 2 it is not only split open, but the two split portions are thrust widely asunder, more or less surrounded by bands of horizontal and oblique white fibers dispersed from other levels by the force of the blow. The whole section shows scattered bands of such misplaced fibers. The columns of Clarke are indicated at *c*.

The two next lower levels are shown successively in Figs. 3 and 4, Plate I. In both of these levels the distortions of the topographical relations of the gray and white matter are more complex. In both sections the same scattered crushed columns of white matter from other levels are present. In Fig. 4 an extra anterior horn on the left side appears to have been thrust up from below. An extra portion of an anterior horn of apparently similar origin is also seen in Fig. 3. Fig. 3 shows also how the dispersed columns of white matter have deflected the posterior sæptum, and have passed across the posterior horns, separating them from the anterior horns.

These drawings show the general characteristics of a moderately extensive bruise of the dorso-lumbar region.

Fig. 6, Plate I, from the same case, shows the appearance of a section where the cord substances have flowed out slightly from the cut surface of the fresh cord. This happens quite frequently and the resulting topographical alterations are quite constant. The anterior horns are liable to be stretched out to

the periphery of the cord, and are surrounded by a wreath of horizontal white fibers, radiating out to the periphery. In this section the right anterior horn is stretched out nearly to the periphery, and a band of white fibers have flowed up from the interior of the cord, and, encircling the distorted horn, pass out horizontally to the periphery. On the left side some extruded white fibers pass out over the pia-matral covering. This appearance at the divided ends of the segments where the cord substances have welled up out of the interior of a soft cord is very superficial. A slight distance below the surface the cord becomes normal.

Fig. 5, from the same cord, some distance above the dorso, lumbar bruise, shows a distortion of the gray anterior horns-the artificial nature of which is betrayed by the horizontal radiating bands of dispersed white matter on either side near the periphery. This section was also taken near the cut surface of a segment where the substance had welled up out of the cord. The changes are similar to those shown in Fig. 6.

A curious porous condition of the right anterior horn is shown, the exact nature of which we have postponed for description in a subsequent paper. This place looks as if a portion of the gray matter had been dissolved out or had been thrust away to some other level in the cord. This condition is an artefact and is frequently met with in bruised cords, especially when instruments have suddenly cut or torn in the gray matter in connection with the bruising.

No changes were found in the central nervous system of this case except the artefacts.

Case II. Fig. 7, Plate II.—The section from which Fig. 7 is taken was loaned by Dr. Ely. Although the case came from one of the foreign laboratories, where the deformity was discovered by accident in the course of microscopical examination, and was then labeled "congenital malformation of the spinal cord," we have taken the liberty of noting the case in its proper light.

This case, like the previous one, is most plainly due to bruising or manipulation, although we have only a single section to judge by, and no notes of the autopsy. The element of bruising is shown by the dispersed and crushed columns of white matter, as in the previous case, and these are indicated by the dotted lines in the drawing. Most of the left posterior horn with its medullary cone and its column of Clarke is dislocated by the force of the blow from its normal site, and is driven out into the anterior fissure.

Some experimental distortions will be shown later, resembling this case quite closely.

Case III. Fig. 8, Plate II.—This section is taken from a case of traumatic tetanus which shows a number of minor deformities of the artificial class. There are no notes about any special features of the removal of the cord at the autopsy. The cord seemed normal grossly except in one place, where the partly detached dura mater was wrapped and tied about the cord, apparently in the fresh condition or during the hardening. In this place the cord is constricted, while above and below it is slightly enlarged.

Fig. 8 is from a section, some distance from the constricted region, taken from the cut surface of a segment, in which the cord matters oozed out while in the fresh condition. In a general way, the appearances resemble those already shown in Figs. 5 and 6, taken from similar places. The anterior horns extend out to the surface of the cord, and the posterior horns are artificially misplaced and malformed. Columns of deviated white matter are also present in the section, but are not shown in the drawing. The general outline of the section also indicates rough handling. Whether the changes are due to the constricting band of the dura mater or bruising at the autopsy, or both, is hard to say.

The appearances of the sections 5, 6, and 8 should be held in mind to compare with a case described by Buchholz as congenital malformation, referred to in Section VI.

Case IV. Figs. 9, 10, Plate II.—This cord was removed by the writer in a case with symptoms of peripheral neuritis at St. Catharine's Hospital, July, 1886, and nothing can be said about the technique of removal except that the instruments were dull and clumsy. The cord was not examined microscopically until some four years afterward, when the contortions were discovered in the review of the laboratory collection.

The distortions in this case are of a slight degree, and are present in the upper dorsal region. In the section corresponding to Fig. 10 the two anterior horns are fused together in one mass, and the gray commissure is very voluminous. The increased amount of gray matter in the commissure appears to have come from some other level rather than from either of the anterior horns. The central canal (*c*) and the columns of Clarke (*x x*), together with the fused anterior horns, have been thrust too far outward toward the anterior surface of the cord. The middle part of the posterior horn on the right side is thinned down to a mere strand and almost separated from the anterior horn, an appearance which seems to have its explanation in the fact that the right anterior horn in its forward displacement has been pulled away from the posterior horn, and the normally thinnest part of the latter has yielded.

A narrow strand of extra gray matter is seen in the left side following the inner border of the posterior horn. The central third of the posterior white columns shows crushed and deviated collections of white fibers not indicated in the drawing.

Fig. 9, from a section a little below the preceding one, shows a very slight distortion of the gray matter, consisting simply in a thinning and partial separation of both posterior horns from the anterior horns at their junction, like the condition of the right posterior horn in the preceding section. Such a condition as this in Fig. 10 or Fig. 9 is liable to be produced by pinching a soft cord with the fingers or feeling of it roughly to test its consistence. The anterior horns do not appear to be displaced forward to any extent. This condition of the posterior horns looks as if it might have been accomplished by gentle bilateral pressure on the cord, which squeezed and thinned them. As the gray matter is softer than the white, it would tend to show the effects of pressure first.

The white matter in this section shows little if any signs of bruising or deflection.

It might be remarked incidentally that the writer has once seen the condition shown in Fig. 10, in a cord which showed no signs of bruising in the microscopical examination, although violence could not be positively excluded. The thinning of the posterior horns occurred in the cervical region in this case to about the same extent as in Fig. 11, and the surrounding arrangement and structure of the cord were perfectly normal. There were no bruises elsewhere in the cord in this case.

So I am not prepared to say that a trifling malformation like the thinning of the posterior horns in Fig. 9 may not occur congenitally without the intervention of artificial causes. But the latter should be thought of first in explaining any deformed condition of the cord, whether trifling or extensive.

To return to Case IV. The probable soft condition of the cord in the hot weather when the autopsy was made, and the transportation for some distance of the fresh cord, mixed up together with the other organs in a common receptacle, should be taken into account in explaining the artificial changes in the gray matter.

There were no lesions in Case IV, and absolutely nothing in the symptoms pointing to the described changes.

Case V (Figs. 12, 13, 14, 15, Plates II and III) relates to a girl, sixteen years old, who had ordinary chorea and died of an intercurrent disease. No notes extant of the details of the removal of cord. *The hardened cord showed no external signs of bruising or pressure*, but transsections through a portion of the mid-dorsal region, about one centimetre and a half in length,

show malarrangement of the cord substances, resulting from bruising at the autopsy or other manipulation.

The section corresponding to Fig. 12 (the lowermost of the series) is a very good example of how a limited column of deflected or displaced white matter may operate on the gray matter. In this instance the deflected column of white matter deeply indents the left anterior horn at the processus lateralis, and fits into an acute angle between the anterior and posterior horns. A still greater deflection of this white column in the same direction would tend to separate entirely the left anterior horn from its connections with the rest of the gray matter.

Figs. 13, 14, and 15, in their order, are the next upward members of the series. This series is exceedingly instructive in showing plainly what has already been spoken of so often—namely, the occurrence of extra bits of gray matter thrust into the section from other levels. In Fig. 13 there is a deficiency of the gray matter, which makes its appearance in the higher levels at 14 and 15.

In Fig. 13 the middle portion of both posterior horns is gone (compare Fig. 10 and Fig. 11, on the right side), and the force driving the absent gray matter to other places is betrayed by two symmetrical, wavy, horizontal hemispherical areas of crushed white fibers, occupying the place of the missing gray matter.

Fig. 14 shows various bands of deflected white matter, a separation of the posterior horns at their middle from the anterior horns, and a portion of the missing gray matter from section 13 interpolated in the middle of the posterior white column, surrounded by bruised white fibers.

Fig. 15 shows very much the same condition, and another tiny lump of the missing gray matter from section 13 is most curiously surrounded by concentrically arranged deflected nerve fibers of the white matter, and is situated in the midst of the posterior white columns. There were no lesions in this cord.

Case V. Fig. 11, Plate II.—This cord was removed from a female adult case of peripheral neuritis by the writer at Charity Hospital, where the use of the chisel and shears are customary, in April, 1889. No details about the cord removal noted. The cord was not diseased in any way.

Fig. 11, Plate II. The bruise (the only one in the cord) is very limited in extent, and lies in the lower part of the eighth cervical segment. It is very much of the same character as that shown in Fig. 12. The outer margin of the left anterior horn is indented by a deviated or crushed bundle of white fibers not shown in the drawing. In Fig. 12 none of the gray matter has been thrust out of the section; but in Fig. 11 a

portion of the horn has apparently been pushed up or down, or both ways, to some other level. Unfortunately, this could not be traced out in other sections, for the rest of the cord in this region has been lost. It will be seen, at any rate, that a group of missing ganglion cells in the left side has apparently gone to some other place.

In addition to these cases, a few instances of artificial malformations due to bruises were found in the cords of animals, in the laboratory collection. These cases are worthy of some note, for two or three cases of bruise artefacts in animals have been described in the literature as congenital malformations of the spinal cord. One of these cases came from a dog dying with rabies. The cord was removed by the writer in 1885, as was supposed, with much care, and without any knowledge of having injured the cord, but a recent examination showed that the cord had nevertheless been slightly bruised—for an inch or so in the upper dorsal region. There was comparatively little deflection of the fibers of the white matter, but in many places the posterior horns were attenuated or misshapen in various ways, and in one or two places were altogether absent on one side or the other, having been squeezed away to other levels. This case should be compared with the case of Schiefferdecker. Many of our sections resemble very closely the supposed congenital malformation in a dog's cord reported by this author (see Section VI). A spinal cord of a sheep showed in the dorsal region a quite similar set of appearances due to bruising or other manipulation.

It is rather a common occurrence to find injuries with the resulting appearances suggesting malformations in the spinal cord of oxen procured at the slaughter-house. The cords are taken out by such crude methods by the workmen that every year, among such cords brought to the laboratory for histological class-work preparation, a certain number show lacerations, splits, and topographical disarrangements of the two cord matters, from bruising. Microscopical examination of the hardened specimens of these cords of oxen in the laboratory collection showed in one or two instances various degrees of deformities from bruising, which need no especial description, but they should be noted because Kronthal (Section VI) instances a case in the litera-

ture with a detailed description of a congenital malformation in the spinal cord of an ox which had been severely bruised.

A rabbit's spinal cord affected with acute myelitis, in spite of the writer's precautions in removing it recently, showed under the microscope deformities from bruising. A doubtful case of an alleged malformation in a rabbit's cord is instanced by Turner (Section VI).

One or two other cases were found in the cord collection, but they show nothing beyond what is already seen in the cited cases. None of the cited cases showed any evidences of pathological processes, and there were absolutely no symptoms in any of them indicating the described changes.

The topographical changes are due to autopsy bruises, or other manipulation, which in these cases was done entirely unwittingly by the pathologist, and I think the cases are valuable in connection with the general subject to show what may be done to the cord, now and then, entirely unawares, even by skilled and experienced pathologists in the course of routine autopsy work.

It will be shown farther on that these bruises can almost always be recognized under the microscope, even if we have no data of the autopsy procedures, or gross external indication of bruising or negligent handling.

Section V.

A DESCRIPTION OF THE RESULTS OF EXPERIMENTAL BRUISING OF THE HUMAN SPINAL CORD.

I. Gross appearances of the bruised cords.

II. Microscopical appearances of the same.

III. Artificial duplication of the spinal cord.

IV. The minute structural artefacts due to bruises of the spinal cord.

The purpose of the work described in this section is to show that the results obtained by experimental bruising of the cord differ in no wise, or in degree only, from the cases cited in the preceding chapter, and also, what is still more important, that these results are the counterparts of the changes described in published cases as congenital defects or heterotopia, in the succeeding section.

During the past two years, as the opportunity of procuring normal human cords at autopsies was presented, some thirty or more of them were subjected to mechanical forces directed in a great variety of ways, and the resulting changes in the cord studied thoroughly with the microscope.

This experimental work on the cord is divided into two kinds:

In one set of cases the cord was purposely bruised in the process of removal from the body.

In another set of cases the cord was removed in perfect condition, and afterward bruised by mechanical forces in a variety of ways.

The first set of cords were injured by exaggerating all of the procedures in the removal of the cord which are liable to damage it as described in Section III. The chisel and the saw were used carelessly and forcibly. The chisel was purposely driven down on the cord, and endeavors were made to drive some fragments of bone or whole laminæ against the dura by the chisel and the mallet. The bone-cutting shears, and levers to pry apart the bones after incompletely sawing them, were also used in a clumsy or forcible way, designedly bruising the dura mater and cord.

In the second set of cords, some were bruised with the dura mater attached, while others were injured after the dura had been removed. Most of them were damaged while within the dura mater, for the latter contributes materially toward the formation of the more complex artificial malformations.

Some of these cords in this second set of cases were bruised by allowing weights to fall on them from shorter or longer distances. Others were struck with different instruments. Small blocks or strips of wood were placed on some of the cords, and the wood struck or tapped with the hammer. In another cord two pieces of tape about an inch apart were suddenly and simultaneously tied down on the dura, etc. Still other cords were pinched with the thumb and finger with various degrees of force—either in a single place in the cord, or at two not far distant places simultaneously. This pinching of the cord with the fingers gives very interesting results, the extent of which can be con-

trolled in a measure by the operator, so as to produce slight deformities, up to doubling or partial doubling of the cord.

The changes in the cord produced by all of these procedures vary so greatly that no general statements can be made as to the uniformity of the alterations produced by any particular mode of applying the force. Hardly any two contortions of the cord from artificial origin look alike. The same procedure applied to two different cords in exactly the same way will not produce constant results as to the microscopic appearances of the distortions. In some cords the extent of the bruise does not appear commensurate with the amount of force exerted, and in other examples the reverse of this appears.

Doubling of the cord is not easy to produce—at least I have not been able as yet to make very perfect duplication of the cord in these experimental bruises, except in two or three cases. The shapes which the cord-substances are liable to assume from the bruising are quite Protean, and there is nothing very definite or constant in the relations of the complex topographical deviations of the cord-matter to the method of bruising. It is even almost impossible, save in one or two forms of bruises, to reproduce any given result.

The presence of the dura mater and the suddenness of the blow may be commented on briefly in the production of these artefacts. It is the presence and position of the dura mater which is largely conducive to the formation of some of the bruises at the autopsy. Were it not for the dura mater, many of the blows from the sharper cutting instruments would simply cut or lacerate the cord without bruising it. But the dura acts as a cushion and distributes the force of the blow so as to disseminate the cord-substances to inappropriate places. If a cord is laid out on the table, and struck suddenly with the chisel, as at the autopsy, with and without the intervention of the dura, the results vary as described. In the first instance the tendency is to bruise the cord without cutting it, while in the latter instance the cord is liable to be incised or cut in two —without much bruising.

After removal from the body, the cord, of course, slips around in the dura, when struck, to a greater extent than

in the body, where both are attached and tense. So the cord on the table may slip away from a tap on the dura mater which it would not escape *in situ* in the dead subject.

If the blow be very sudden, the tendency is to scatter the cord substances about more extensively and grotesquely than in slow, deliberate pressures. If the cord be slowly pinched with the thumb and finger, the white and gray matter may be driven above or below, each remaining in its proper place. The cord becomes larger above or below the cincture, the white matter is more voluminous, the fibers run in abnormal directions, and the gray matter tending to remain in its proper position is larger, and pretty uniformly so in all of its different parts. The result produced is a sort of artificial numerical hypertrophy.

But if the same amount of force expended in this slow manual cincture be concentrated in a sudden sharp impact, the result is altogether different; the gray matter and white matters are detached and scattered about in all sorts of directions.

Of course, all these various modifications of the application of the force are in turn modified by the consistence or freshness of the cord, and this produces correspondingly different phases of deformities. Thus, it may be said that a single blow at the autopsy on the dura mater may produce any of the infinite varieties of distortions, from a slight asymmetry up to a condition in which a whole portion of a segment is shifted bodily to another level, giving a doubling of the cord.

I. Description of Gross Appearances of Bruises of the Spinal Cord.

The blow may leave no gross changes. Perhaps the natural elasticity of the cord membranes or the cord tissues may reduce the cord to its normal contour to some extent after the bruise. A bruise without gross traces in the fresh cord will always show with careful microscopical examination, and sometimes to a very considerable extent in the dispersion of the cord substances. In other cases there are the extensive changes shown in Plates IV to VI, which cover a sufficient variety of phases to make the matter clear.

Case I. Fig. 16, Plate IV.—This cord was removed from

a horse dying with azotorrhœa—a disease in horses often manifesting symptoms of paraplegia apparently of a toxic nature. The cord was removed by the writer some years ago, prior to commencing these studies. The autopsy was made in hot weather, twenty-four hours after death, and presented so much difficulty in the use of the ordinary instruments that finally an axe was used to complete the cord removal. The lower dorsal and lumbar portions of the vertebral column were chopped out entire, and then split open with the sharp axe.

At one point in the lower dorsal region there was a circumscribed, fusiform swelling of the cord with diminished consistence and a trifle of flattening. Fig. 16 shows this appearance, and I had the plate drawn from the fresh cord for intended publication, under the impression that softened swelling was a focus of acute myelitis, which corresponded well enough with the symptoms, as the horse had more or less complete paraplegia. Not until quite recently was the cord examined microscopically, when it was positively seen that the appearances were due to bruising and that there were no lesions in the cord.

The cord must have been bruised by a slanting blow of the axe in splitting open the vertebral column. While the gross external changes are considerable in this case, the topographical alterations of the gray and white matters are only of a very moderate degree. This is a very striking exception to the rule—which is, that such striking gross changes as are present in this case correspond to extensive topographical deformities in the sections. The only displacements of the cord substances, however, in this case are limited to slight dislocations and disfigurations of the posterior horns. The customary deflections of the white fibers are quite limited and in many sections entirely absent.

The most interesting feature of the case is the presence of rather large, ragged-walled cavities of fairly considerable vertical extent in the posterior white columns. In one region the cavity takes up nearly the whole of the posterior columns. These cavities are part and parcel of the effects of the bruise, but to an observer forgetful of the effects of bruising they might suggest the lesions of syringomyelia, or appear like preformed cavities. These cavities had no

lining membrane, and scattered about diffusely in their ragged, lacerated walls were some extravasations of red blood-cells, also of artificial mechanical origin. The cases of Köppen—discussed more fully in Section VI—should be referred to in connection with these artificial cavities. Köppen instances two cases of what he terms "formations of cavities in the spinal cord of acute origin." While not venturing to question too positively the preformed nature of the cavities in the author's cases, the writer would suggest the perfectly possible origin of cavities, together with the extravasations of blood in their walls, as due to bruise artefacts.

To return to our own case, the cavities seem to have been produced by mere lacerations or a rending apart of the white matter by the force of the blow. If not produced in this way, they may have been caused by the transportation to other levels of crushed masses or detritus of white matter which have left a cavity behind (see Fig. 24). The borders of the cavity consist of perfectly normal white matter and are remarkable in showing no mechanical dispersions of the white fibers so characteristic of bruising. Such cavities occur not infrequently in bruised cords, but rarely take such a deceptive nearly circular or oval form and position in the center of the cord as in the present case, but most frequently assume the shape of purely mechanical splits or clefts. These artificial cavities in the white matter have some resemblance to the "porous condition of the gray matter" already described, but they appear to be produced in a slightly different manner.

Figs. 17 and 18 (Case II, Plate V) are taken from two different places in the dorsal region of the same cord. Both of the bruises were produced by sudden compression with the thumb and finger, with the intention of reproducing the artificial doubling of the cord in the two published cases of Seguin and Fürstner and Zacher (see Section VI).

In Fig. 17 there are two bruises—an upper and a lower one. In the lower one a portion of the cord, principally white matter, has broken through and has been squeezed out of the pia matral covering of the cord. The red streak in the extruded mass, supposed in the fresh condition to be gray

matter, was found in subsequent microscopical examination to be a distorted blood-vessel surrounded by some artificial hæmorrhages. The second upper bruise is indicated by a circumscribed globular lumpy swelling out of the cord contours. The extruded mass below was soft and pasty in the fresh condition, like the condition of the white matter in a softened normal cord in late autopsies in warm weather. The remarkable feature of these two bruises is that the cord does not show the usual girdles of deficiency to compensate for the increased volume of the cord at the bruised regions as distinctly as usual. As a rule, where the cord is struck there is a dent or cincture, and a swollen lump of the dispersed cord-matters is found above or below. But the correspondence of volumetric relations in the bruises does not appear at all as frequently as would be expected. Very often the cord may show the lump-like appearance as in the upper bruise of Fig. 17, and yet above and below the lump the cord may seem very nearly normal in its volume. The sense of touch, however, will sometimes give a better appreciation of the deficiency above or below the lump in the cord than the eye.

In the portion of the cord represented by Fig. 18 the pressure by the thumb and finger was applied simultaneously on either side of a given point; the pressure in this instance was rather more deliberate and gentle than in the portion shown by Fig. 17. The extruded mass here lies on the anterior surface of the cord, and consists exclusively of white matter; there is also a slight fusiform swelling of the cord at this point, while above and below, the cord is slightly smaller than elsewhere in the same region. (It was impossible for the artist to show this diminution of the cord volume well above and below the swelling in the position the cord was placed. To show this, another picture of the lateral face of the cord would have been requisite. The pinching was applied to the cord bilaterally). The plate shows well enough what has happened from the pinching to dispense with further detail. Sections from both of these cords will be described in connection with the microscopical appearances of the experimental bruises. Fig. 62, Plate XV, is from a section through the extruded mass in Fig. 17, and Fig. 63, same plate, corresponds to Fig. 18.

Case III. Figs. 19 to 20, Plate VI.— The bruise in this case was effected in the purposely careless removal of a firm cord in cold weather, from an ordinary case of phthisis, at Charity Hospital. The chisel and the shears were used freely. The chisel had shoulders to arrest its entrance into the spinal canal, but it was driven down through the lumbar laminæ with a sharp mallet-blow, with the result of impacting a broad splinter of bone into the vertebral canal. To get rid of this, the bone-cutting shears (a particularly powerful and awkward pair) were used. Between the action of both instruments, although the dura mater showed no indications of it, the cord was damaged as depicted in Fig. 20, which shows a depression in the cord, and corresponding to it an extrusion of cord substances from the depressed place. The bruised place was a trifle above the commencement of the cauda equina. A doubling of the cord was sought in this experiment.

The cord in Case IV was taken at the same hospital, in a similar manner, to reproduce doubling of the cord. The chisel alone was used, and the laminæ in places, without being sawed, were severely hammered with the chisel and heavy mallet with the result shown in Fig. 19. The plate tells its own story well enough without further explanation. The remarkable thing about the cord is the extrusion of the cord matters clear through the dura mater in the upper bruise. This was probably furthered by the tension and attachments of the cord and membranes while in the vertebral canal. The bruises in this cord are confined to the dorsal region. In this instance a remarkably perfect duplication of the cord was produced. One portion of the cord was telescoped down sidewise over another portion, and the result is shown in Figs. 64 and 65, Plate XV, drawn from sections through the cord and the extruded portion alongside of it.

These last cases are examples of extraordinarily severe bruises. The lesser contusions show a mere diffuse irregularity in the exterior contour of the cord. Or there may be a single very circumscribed dent or dimple in the surface, or a minute prominence or pimple-like projection. Still, other cords show little mounds and depressions on the surface, multiple or aggregated in their distribution. Some-

times there are minute multiple extrusions of the cord substances on the surface of the cord (see Fig. 23, Plate VII), some of them fused together in larger or smaller masses.

The consistence of the cord is diminished at the seat of the bruise corresponding to the severity of the blow. If the bruise is severe and the superficial covering of the cord is not ruptured, the cord feels soft and pulp-like within the wrinkled or partly collapsed membranes. If a portion of the cord bursts through the membranes in the bruising, it is liable to be soft and pasty and yellowish-white in color. Sometimes the extruded masses show larger and smaller artificial hæmorrhages in their substance (Fig. 18). If a portion of the cord is dislodged to some other level, and yet does not burst through the membranes as in Fig. 18, *a*, it produces a larger or smaller lump which bulges out the contour of the cord. In such places (Fig. 18) the consistence of the cord is increased, and the superficial neuroglia coat is tense and hard. A normal cord which has been bruised extensively feels somewhat like a cord which has been softened by acute myelitis.

In the hardened cord these bruises also show very well, and their external topography can be more conveniently studied. The slightest extrusion of cord substances on the surface should lead to the suspicion of a contusion.

While hardening in Müller's fluid, the deflected bands of white matter in bruised cords assume the color of the gray matter. They appear as little gray spots or diffuse areas scattered about over the surface of the transverse section, and this occurrence, often coupled with dispersed fragments of gray matter, gives a spotted appearance to the cut surfaces of the cord while in Müller's fluid or in the subsequent alcohol immersion. These strikingly distinct circumscribed or diffuse spots often look so much like lesions that the observer, in reconnoitering the specimen by transverse incisions during the hardening stages, is liable to consider them foci of softening or disseminated myelitis.

In Plate VII the drawings were made from the hardened specimens, and show some additional points about the macroscopical appearances of bruises of the spinal cord.

Fig. 21 was drawn in 1885 from a specimen removed at Bellevue Hospital, from a case with symptoms of peripheral

neuritis. The writer was completely misled by the gross appearances, being then ignorant of the numerous artefacts caused by accidental autopsy bruises of the spinal cord. The lesions of this disease were less well understood at that time, and in searching for changes in the spinal cord the appearances at *a* (Fig. 21) were described in detail in the notes of the macroscopical features as a tumor or solitary tubercle involving the third lumbar segment, which was supposed to agree with the symptoms. The little lump-like protuberance was really caused by bruising the cord; a portion of the white matter was thrust out of the cord, and this distended and pouched out the superficial neuroglia coat so as to resemble a small neoplasm. An exceptional feature about the case is that there was no deformity of the gray matter anywhere in the cord, or even much disturbance of the white matter, excepting the portion thrust out into the protuberance. Generally the force of a bruise sufficient to extrude the marginal white matter in this way also distorts the gray horns considerably. Such a limited circumscribed effect of a bruise as in this case is very uncommon.

Fig. 22 is another instance in which the results of a bruise simulate the appearances of a tumor of the cord quite closely. The cord was bruised experimentally, and a considerable portion of the cord substances have been thrust out through the lateral surface of the cord. The lower sectional surface of the drawing shows a deficiency in volume corresponding to the extruded portion. The cord was bruised with the intention of producing a duplication of the organ, which was but partially successful, as shown in Fig. 66, Plate XV, drawn from a section of the specimen.

The cord in Fig. 23 has been very severely bruised, partly in the autopsy procedures and partly by manipulation after its removal. In two places the cord is quite thoroughly flattened and has had its substances pushed both upward and downward, and forms two corresponding swollen regions. *a* represents the upper dorsal and *b* the lower cervical region. The specimen shows especially well how larger and smaller portions of the cord substances (principally white matter) have burst through the external coverings of the cord, and appear as multiple wart-like ex-

crescences. Duplication of the cord was produced in both of the swollen regions. See Figs. 67 to 69, Plate XV, from the upper swollen region, and Figs. 70 to 75 from the lower swelling.

Fig. 24 is exceedingly interesting. This drawing gives a good idea of the actual movement of a deflected column of the two cord substances (principally white matter) from one level to another, and how the presence of the transported column in the new level distorts the topography of the cord. In this instance the deflected column splits open the left anterior and posterior horn, and fits into the cavity as a plug. The left-hand segment of the cord was struck with the ulnar side of the hand while lying on the table. The cord was hardened without incisions for some weeks in Müller's fluid, and while yet pliable was patiently dissected and opened like a hinge, so that the plug of dispersed white matter was exposed and pulled out of the split in the gray matter. This drawing, then, shows one of the ways in which deceptive cavities may occur in bruised cords. If in handling the cord after the bruise such a plug should be moved back again whence it came, or should shrink a little, a cavity would appear in the sections. The cavities in the horse's cord in Fig. 16, Plate IV, may have been formed in some such way as this. The sections from this specimen show nothing of especial interest and they have not been pictured. If the plug had been left in place, sections from the right-hand segment of the cord would look quite similar to Fig. 1, Plate I.

Taken all together, these gross appearances are most characteristic. There is no other condition of the cord which looks like them, and it is difficult to understand how they should have been mistaken for congenital deformities or heterotopia. It must be that some of the published cases of bruises mistaken for congenital malformations have been described from microscopic appearances by observers who did not have the opportunity of seeing the fresh gross appearances at the autopsy. *It may be repeated that very often quite extensive artificial displacements of the cord substances never show at the autopsy at all, or alter the external gross appearances of the hardened cord.*

II. Microscopical Appearances in Experimental Bruising of the Cord.

The microscopical appearances of the sections from these experimental bruises vary so greatly and are so difficult to convey an intelligent idea of by mere description, that it has been thought best to replace the text of this section largely by drawings. The intention is to show counterparts of the microscopical characters described in the published cases, and to show a sufficient number of various phases of the results of bruising to convince the reader that the cases in the preceding as well as the published cases in the succeeding chapter are simply artefacts of the spinal cord.

Case I. Figs. 25–30, Plate VIII.—These sections were taken from a fairly firm, carefully removed normal cord upon which a flat twenty-gramme weight was allowed to fall from a distance of twelve to eighteen inches, with the result shown in the figures. The cord was protected by the dura and laid out on a table. The weight fell on the cervical enlargement.

In Fig. 25, the lowermost of the series, the right anterior horn is absent, while the left anterior horn is deeply indented. The absent right anterior horn begins to make its appearance in the higher levels in Figs. 29 and 30. Some deflected white matter has been thrust outside of the periphery in Fig. 25. Another feature in this section is the deflection of the white fibers by the connective-tissue strands of the cord. In the right lateral column several bands of fibers pursue contrary and devious directions, caused by deflections, according to the directions and course or strength of the connective-tissue sæpta.

Figs. 26 and 27 show very much the same conditions as in Fig. 25. The left anterior horn is deeply indented and the right anterior horns become more voluminous and are very much disfigured. In Fig. 26 radiating, brush-like deflected white fibers pass out toward the periphery from the anterior horns. In Fig. 27 the areas of mechanically dispersed fibers of the white matter are indicated by the dotted lines. In Fig. 28 the right anterior horn becomes considerably larger and sends off a spur-like process into the lateral column.

In Fig. 29 the most striking feature is a dislocated or extra fragment of gray matter on the right side which has been thrust up from the lower levels. Fig. 30 shows this extra fragment of gray matter with its ganglion cells, fused with the right anterior horn, which is indented by a deflected band of fibers in the white matter.

In all of these sections the artificial changes in the white matter merit attention, particularly the changes in the directions of the dispersed nerve fibers relative to the posterior sæptum and the two connective-tissue strands and vessels bounding the column of Goll. Fig. 25 corresponds to the first dorsal segment and Fig. 30 to the upper surface of the eighth cervical segment.

In Fig. 31 the indentation of the right anterior horn is still more pronounced and causes a singular spur-like projection of gray matter with ganglion cells, from the middle of the posterior horn. This figure again shows exceedingly good examples in the posterior columns of how, when the white fibers are driven against the stronger connective-tissue sæpta, they are deflected in various ways.

Just behind the spur-like projection in Fig. 31 at *a* will be noticed a mass of deflected white fibers different in appearance from the other deviated fibers, which pass about in wavy parallel lines for the most part parallel to the plane of the section. The mass alluded to is different, in the fact that the fibers are crushed together and disintegrated and give the appearance of an amorphous or coarsely granular mass. A similar group is seen at *b*. This behavior of the fibers, when crushed together instead of being simply deflected while retaining their structure, will be frequently seen in the drawings from the other sections. These crushed bundles of fibers are less frequently observed than the other variety of deflected fibers.

Case II. Figs. 32, 33, Plate IX.—The cord in this case was struck in the upper lumbar region with the ulnar side of the hand while lying on the table stripped of its dura mater.

Fig. 32 shows an absence of nearly the whole gray matter on the left side, while, what is very remarkable, the right anterior and posterior horns are perfect. Fig. 33 shows a similar condition. The missing gray matter was not traced to its misplaced destination in this case.

The appearance of the left segment of gray matter in Fig. 33 is remarkable. The shrinkage of the gray matter from the pressure of the blow is so uniform in all directions that an atrophic condition would be suggested were the observer not cognizant of the manifold topographical artefacts of bruises of the cord. The condition of the shrunken gray segment here looks a little like the condition found in the adult cord after intra-uterine amputations, or the apparent hemiatrophy suggests the condition in an adult after poliomyelitis anterior. In looking at Fig. 32 we can readily understand how the whole segment of the gray matter on one side might be transported to another level, and result in the presence of three segments of gray matter. Just such a condition as this misleads writers to describe the appearances as a congenital partial duplication of the cord. Both of the sections correspond to the second lumbar segment.

Case III. Fig. 34, Plate IX.—A hard normal cord was tapped by the autopsy chisel, while lying stripped of its dura on the table, in the mid-dorsal region. A narrow band of deflected white fibers have cut off the tip of the left anterior horn and a very near approach to the same thing is apparent in the right anterior horn.

Case IV. Fig. 35.—Cord treated similarly as in Case III. In this contusion may be remarked the lengthening out of the right anterior horn, the flowing out of the deflected white fibers beyond the periphery of the cord, and a small dislocated portion of an anterior horn from some other level.

Case V. Fig. 36.—Manual torsion applied to the naked cord. This shows the result plainly enough to omit description.

Case VI. Figs. 37 to 39, Plate X.—A fairly firm normal cord was gently pinched with thumb and finger in the mid-dorsal region. Figs. 37 to 39 constitute a series through this contusion. In Fig. 37 the commissure and anterior horns are absent and there is much diffusion of the white fibers in a horizontal direction. In Fig. 39 the deficiency in Fig. 35 appears as extra gray matter, causing the appearance of a third anterior horn springing from the gray commissure between the other two horns rightfully

belonging in the section. Fig. 39 shows very well on the left side the production of the minute multiple excrescences of white matter on the surface of the cord referred to in the description of the gross appearances of the experimental bruises.

Case VI. Figs. 40, 41, Plate XI.—The cord protected by the dura was intentionally damaged with the saw in its removal. In Fig. 40 a spur like projection of the left anterior horn has been produced by a shallow depression in the gray matter. In front of the left horn are two isolated bits of gray matter containing ganglion cells lying surrounded by deflected tracts of white matter which are indicated by the dotted areas. In the column of Goll, corresponding to the position of the sciatic field, is a very compact mass of crushed fibers which simulates the appearance of a sclerotic patch with carmine staining. The simulation of a sclerotic patch is quite deceptive, and these crushed areas of white columns have probably been described as sclerotic or other diseased patches.

In Fig. 41 the projecting spur is broken up into three small bits of gray matter, and this figure also shows how the spur was formed at the expense of the gray matter of the left anterior horn. This figure also shows an oval patch of wavy white fibers grouped on either side of the posterior sæptum, and this would not simulate sclerosis, as in the other section, as the outlines and course of the fibers are distinct. Attention may be called to the singular circumscribed deflection of white fibers on either side of one of the smaller stiff connective-tissue sæpta in the left lateral column near the middle of the elongated posterior horn.

Case VII. Fig. 42.—This section was also taken from a cord experimentally bruised in its removal by the osteotome at Roosevelt Hospital, and needs no special description. It is simply another of the many attitudes which the cord substances may assume when bruised mechanically. In the left lateral column two sets of deviated white fibers are most curiously parted on either side of a short connective-tissue sæptum, like the hairs of the head.

I am unable to determine at present just how this singular disposition of the deflected white fibers about the connective-tissue sæpta and blood-vessels is to be accounted

for. In some places it looks as if the oscillation of the sæpta themselves, from the shock of the blow, had deflected the white matter, yet in other places the appearances are such as to indicate that the white column had been moved first and had then run up against some sæptum in its path with an ensuing change in the direction of the deviated column. Probably both of these agencies work hand in hand in many instances. At any rate, the presence and arrangement of the sæpta and blood-vessels in the white matter play a very important part, and account for some of the bizarre forms in the arrangement and distribution of the deviated white fasciculi.

Quite frequently the displaced white fasciculus is moved forcibly enough to break through the coats of strongest sæpta of the cord, such as the sæptum posterius (see Figs. 43 to 47).

Case VIII. Fig. 43.—This cord was bruised by cinctures, effected by tying suddenly broad pieces of tape around the dura, and Figs. 40 to 42 constitute a series showing the changes a slight distance above one of the moderately severe cinctures in the mid-dorsal region. This is one of the most frequent alterations which ensues from bruises of all kinds—viz., an eruption of white matter in the middle of the cord, bursting through the gray commissure and thrusting asunder the two anterior horns. One or another modification of this result happens very often indeed.

Fig. 43 depicts the slighter grade of intensity of this process of median rupture. A slender band of white fibers, appearing from some other near level, has traversed the posterior sæptum, and tends to cut the right anterior horn in two.

Fig. 44 shows a similar result, except that the deflected white fasciculus has passed in another direction, has burst through the gray commissure, which in Fig. 45 is partially disrupted by a continuance of the same process. The rupture of the centrally situated white matter has also thrust the two anterior horns apart to a considerable extent in this section.

In both of these sections (Figs. 43, 44) the column of diverted fibers of the white matter is quite limited, and there is a correspondingly small degree of deformity in

the gray segments. If the reader will review the preceding sections he will observe that the greater the amount of dispersion of the white fibers, the greater is the deformity of the gray matter. Thus, as a general rule, excessive deformities of the gray matter go hand in hand with extensive deflections of the white matter.

Case IX. Figs. 46, 47, Plate XII.—Figs. 46 and 47 belong together and are taken from serial sections of a cord intentionally wounded at the autopsy. In Fig. 46 the left gray segment is separated from its fellow and curved out laterally toward the periphery of the section. Two fragments of extra gray matter are seen in the region between the two separated gray segments, and are surrounded by a mass of deflected white fibers which are cut obliquely. The source of the extra bits of gray matter can be seen in Fig. 47, where there is a corresponding deficiency of gray matter on the left side.

Fig. 48 needs no special description, for it is isolated from its fellow-sections, which show the destination of the dislocated anterior horn on the right side. The wavy lines clustered about the region of the dislocated horn show a very faithful image of just how these horizontally deflected nerve fibers of the white matter look in the section.

Figs. 49 to 51 correspond to a series of sections from a cord purposely bruised in its removal. The reader should mentally reverse Fig. 49 from right to left so that it will correspond properly with Figs. 50 and 51. One of the anterior horns in Fig. 49 has been nipped off at its junction with the gray commissure by a hemispherical mass of deflected white fibers passing horizontally in the section. The absent horn appears again as two fragments in Fig. 50, which are united in one mass in Fig. 51. The artificial changes in the white matter in all three sections are very typical.

All of the sections in Plates XI and XII are from the dorsal region except Figs. 40 and 41, which are from the eighth cervical segment.

It may be remarked here that the aberrant cord substances, particularly the gray matter, which can be traced more readily than the white, are sometimes dispersed to considerable distances in the cord. Roughly speaking, the

aberrant or dislocated gray fragments may be caused to travel a distance in the cord from one, two or three millimetres to a good portion of a centimetre. No notes, however, have been made of accurate measurements in reference to this matter.

Figs. 52 to 55, Plate XIII.—These drawings are made from sections selected at random from a number of artificially bruised cords about which there is nothing especially noteworthy in details of the method of bruising, except in the sections corresponding to Figs. 54 and 55.

The two cords from which these sections were taken were prepared by placing portions of the posterior archway of the vertebral column on the cord lying on the table within the dura, and manipulating the bones with the chisel and hammer.

Fig. 52 shows no especially new features about the gray matter except in the dislocation of the right anterior horn, but the condition of the white matter in the anterior portion of the section deserves inspection, for it shows how the mechanically dispersed nerve fibers appear when passing through the section obliquely instead of horizontally, as is usually the case. Here the dispersed nerve fibers are in the act of bending over to seek some new level in the cord.

What has happened in Fig. 53 is shown plainly enough in the drawing to omit further description. The changes in the white matter in the center of the cord are not represented.

Fig. 54 is instructive in demonstrating how nearly all of the gray matter may be driven away from a given level. In extensive bruises, where a portion of a whole segment is shifted within or partly within the cord coverings to another level producing doubling of the cord, the deficient portion may be so deprived of the cord substances as to present hardly anything but the collapsed cord membranes in section. Such a complete expulsion of the gray matter to another region as shown here is liable to produce more or less complete duplication of the cord in the new levels, and Fig. 54 is the sort of picture we often see above or below artificial duplications of the cord.

The striking feature in Fig. 55 is the very extensive

diffusion of the artefacts in the white matter; there are no normally situated white fibers in the whole section; the entire territory of the white matter is occupied by deflected fibers, and even the connective-tissue sæpta are broken and distorted. Fig. 56 will be described among the artificial duplications of the cord in the succeeding pages.

III. Artificial Duplication of the Spinal Cord.

After having described the simpler forms of bruises of the cord, which constitute the majority of the cases published as heterotopia of congenital origin, it remains for us to study the more complex results of bruises producing artificial doubling of the cord, for these have also been described in a few instances in the literature (Fürstner and Zacher, Kronthal, Feist) as congenital malformations.

Injuries to the cord at the autopsy seldom result in duplication of the organ. The direct or indirect blows from the instruments almost always produce the minor forms of deformities; if the blows be very violent, the cord is lacerated or mutilated, but not often doubled. In fact, any perfect degree of duplication is very difficult to produce by any sort of experimental means. Yet, occasionally in removing the cord the operator may strike the organ in such a way as to cause doubling over a limited region without apparently having in mind the least possibility of the entirely artificial production of such a result.

There is not any very distinct line of demarkation in these bruises of the cord between the lesser topographical alterations and complete duplication of the organ. They merge into each other. The several complicating factors incident to bruising of the cord at the autopsy—such as the several degrees of the force of the blow, its direction, the driving down of larger and smaller fragments of bone on the cord, the consistence of the cord itself, etc.—are so variable that, when artificial doubling of the cord is effected, the topographical artefacts in the sections are not at all constant. The changes are just as irregular as in the lesser grades of bruise already studied.

Thus there are several degrees of doubling of the cord due to bruises; the operation of the variable factors incident to bruising may result in dislodging only a small or

incomplete portion of the cord from one place, and, fusing this dislodged portion with the periphery of the cord at another level, may produce only a partial doubling. On the other hand, the bruise may shift a complete segment and telescope it within or over the periphery of the cord, above or below, and produce quite a perfect duplication of the organ. Between these two forms of duplications—the incomplete and the more perfect forms—there are all sorts of intermediate gradations.

The different writers meeting with these intermediate forms of doubling, and mistaking them for congenital malformations, describe them as "rudimentary cords" (persisting and remaining fused to the primary cord), or "partial or incomplete duplications of the cord of embryonal origin."

The reason that perfect duplication of the cord from bruising—that is, more or less perfectly formed cords lying side by side or fused together within the periphery of the cord—is so difficult to produce by experiments or accidentally in routine autopsy work, is because the shifted segment of the cord becomes so badly disfigured or mutilated in its dislodgment and transit from one level to another. The cases of Fürstner and Zacher and Seguin (see Section VI) are remarkably perfect results in duplication from autopsy manipulation. The incomplete forms of duplication, however, are produced not infrequently at autopsies, and are very readily produced experimentally.

The gross appearances of these cord duplications tend to resemble the features shown in Plates V and VI. Sections from the cord in Fig. 19 showed a very perfect form of duplication. Sections of the other cords in these plates do not show complete duplications, but merely extrusions of the white matter. The cord is always more voluminous at the seat of the duplication, and above or below will be found a deficient, collapsed, or flattened region, corresponding to the transported segment. Generally in these duplications the dislodged segment bursts through the neuroglia sheath of the cord and is pushed up or down over the surface of the organ, while still attached to the original site by a broad or slender pedicle (Plates V and VI). More rarely a segment of the cord may be telescoped over another, without rupt-

uring to any great extent through the outside covering of the organ (Plate VII, Fig. 23). Duplications of this kind are nearly always very circumscribed. They seldom occupy more than one half to one centimetre in vertical extent, and often but a few sections can be cut through them.

A duplication of the cord may be produced by a single blow over a limited region; but the most successful way of producing duplications is to bruise or compress the cord in two places either side of a given point. In such an instance the doubled region has a fusiform shape, with blunt, conical, or flattened ends (see Fig. 23). Very likely some of the artificial duplications recorded in the literature were produced by striking the cord in two different not far distant places.

The following plates show the best results of duplications in very many cords experimented with. While we have not been able to exactly reproduce the appearances of the sections in the cases of Fürstner and Zacher, Seguin, and Feist—for it is excessively difficult to reproduce the given appearances of any bruise, or even to get the same result from like bruises of two different cords—yet these experimental results approximate the features of these cases comprehensively enough to demonstrate that these "rudimentary cords" and "partial" or "complete duplications of the cord," described in the literature as malformations, are nothing but bruises.

Fig. 56, Plate XIII. This cord was pinched with the fingers, either side of a given point. Before pinching the cord a little slit was made in the superficial neurogliar covering, on the postero-lateral surface, to let the dislodged cord matters escape.

The section (from the first or second sacral segments) shows a failure in duplicating the cord rather than a good result. Quite a large volume of nerve fibers have been squeezed up from below and run out horizontally from the posterior column beyond the periphery of the section. The gray matter in the extruded portion is almost entirely composed of the gelatinous substance of Rolando, which, near the periphery of the cord, is split up into anastomosing trabeculæ by deflected columns of white matter.

Figs. 57 to 61, Plate XIV, constitute a series through a

bruise of the cord which produced more or less complete duplication. The cord—one of rather diminished consistence; autopsy forty-eight hours after death, in warm weather—was injured by driving down fragments of bone on it while it lay in the vertebral canal, within the unopened dura, after the posterior vertebral archway had been removed.

Fig. 57. In this drawing there are four anterior horns. There is a complete section of the cord, and fused on its anterior surface is a portion of the cord from some other level, which contains two anterior horns. In the complete section the anterior horns are curiously lengthened and curved, and are surrounded by dislodged fibers of the white matter. The piece of gray matter indicated by *y* is difficult to identify. It looks like a part of a posterior horn.

What has happened here is, that a portion of the cord from one level has been forced down along the anterior surface of the cord to another level, where there has been a partial fusion of the two portions. The places marked *x x x* show some of the minute structural artefacts of the white sometimes produced by bruising. In these areas the fibers have not been thrust out of their course; they are still in their proper position, but have been squeezed together and have been changed structurally, so that with the low power, and in their behavior with carmine, they resemble areas of sclerosis or secondary degeneration.

Fig. 58, the next of the series, shows a still greater extent of the dislodged segment, and how it has been telescoped into the anterior fissure, spreading apart the lips of the fissure, and the anterior horns at another level in the cord. The dislodged portion has an anterior fissure as well as a set of anterior horns, with a dislocated commissure. The posterior horns belonging to the extra-anterior horns seem to have been left behind at their proper level. At *x* is shown again one of the minute structural artefacts in the gray matter from bruising—viz., a rarefaction or mechanical disintegration of the gray matter, so that a porous condition resu'ts, which stains faintly.

Fig. 59. This section shows only a slightly different condition from the preceding one. The dislodged segment is still impacted in the anterior fissure, and the two extra-

anterior horns are fused together in a U-shaped mass. Both arms, *a c*, as well as their junction, *b*, contain scattered ganglion cells. At *d d* are some deflected bundles of nerve fibers which are rather unusual, in the fact that, although grouped together, they are isolated, and occur in such small, closely approximated bundles. The majority of these bundles (*d d*) run obliquely through the section; *f f f* indicates some masses of white fibers which are not altered topographically, but structurally, as described in Fig. 51, *x x x*. A porous or rarefied condition of the gray matter, almost amounting to an actual cavity, is seen at *e*.

Fig. 60 shows, with a slight modification, the same appearances as in Fig. 59. The new feature in this drawing is the extra portion of gray matter united to the tip of the left anterior horn in the complete section. This seems to be a third extrinsic anterior horn, and not a lengthened-out portion of the horn properly belonging in the section. So at this level of the bruise there are portions of five anterior horns. In the right-hand arm of the U-shaped portion of gray matter in the dislodged segment will be noticed a feature which sometimes occurs in a bruise—viz., that the filamentous processes radiating out from a displaced horn become rubbed off or partially disappear in the transit to new levels. In this way a dislocated horn sometimes has a smooth appearance as to its bounding surfaces. As in the previous sections, the dislodged segment is crowded into the anterior fissure of the complete section, but is quite intimately fused with the rest of the cord at the new level.

Fig. 61, the final member of the series, shows the fusion of the U-shaped mass with the gray matter at the level of the cord, where the mass has been telescoped. The four anterior horns have been joined together, two by two, at *a* and *b*, which gives the section a most curious configuration. At *a* are some horizontally deflected nerve fibers of the white matter which give the deceptive appearance of commissural fibers passing between the two fused anterior horns (compare with Kronthal's alleged case of malformation of spinal cord of an ox in Section VI). Some bundles of white matter which have been thrust up through the gray matter are shown at *d*. This mechanical intrusion of columns of white matter into the gray matter occurs quite

frequently in bruises, but the writers on heterotopia misunderstand them, and call them "anomalous bundles," supposed to be misplaced congenitally (see Feist's first case, Section VI); *e* denotes a rarefied place in the gray matter, and *f f f* have the same significance as in Fig. 59, and as *x x x* in Fig. 57.

Fig. 62, Plate XV, is from a section through the lump of extruded cord substances shown in Fig. 17. The bruise has not doubled the cord, as was intended; the portion dislodged by the bruise consists almost entirely of white matter. The two indentations on the right-hand part of the section containing cross-sections of the nerve roots indicate the boundary line between the extruded portion and the cord itself. The deflected nerve fibers in the left posterior column describe a U-shaped arc, and at *x* and *y* these fibers turn vertically out of the section after pursuing a horizontal course across the posterior column. The right posterior column is occupied by obliquely diverted fibers (see V); *z* shows again the effects of connective-tissue sepia in modifying the course of the deflected fibers.

A very important thing in this section is the occurrence of minute multiple hæmorrhages induced mechanically by the bruise. The force of a bruise is sufficient sometimes to burst the walls of the smaller blood-vessels and disseminate their contents about into the cord substances, sometimes in the white matter, but generally most extensively in the gray matter. The drawing shows but a few of the larger artificial hæmorrhages; they are indicated by the deep-black spots in the right interior horn (compare with the hæmorrhages described by Buchholz in his first case in Section VI). At *w* are indicated a group of fibers in the white matter which are not dispersed or altered topographically, but are changed in their minute structural details so that they stain in a slightly different way from the surrounding fibers.

Fig. 63 is from a section through the bruised part of the cord shown in Fig. 19, Plate V, and needs no detailed explanation other than that it is a very faithful representation of how these horizontally displaced nerve fibers of the white matter appear in a section.

Fig. 66, corresponding to the second lumbar segment, is

from the cord shown in Fig. 22, Plate VII. The extruded portion consists of both gray and white matter, and is connected to the cord by a slender pedicle, through which dispersed white fibers have flowed out into the lateral mass. Sections from bruises, resembling this drawing, have been pictured as showing a "rudimentary cord" attached to the primary cord (see plates of Kronthal's first case).

Figs. 64 and 65. These drawings show a very perfect form of duplication of the cord. The sections were taken through the extruded portion of the cord in Fig. 19. The drawings show two portions of the upper dorsal cord lying side by side. In Fig. 64 the dislodged portion is unattached to the cord save by a few shreds of pia mater, while in Fig. 65 a narrow pedicle attaches the misplaced segment to the cord itself. The whole area of the white matter is occupied by deflected fibers running in various directions, but they are not shown in the drawings.

This is the most perfect duplication of the cord which we have yet succeeded in producing, and it is important to note that the sections, although from another region in the cord, are fully as perfect as in the case of Fürstner and Zacher (see plate in Section VI), which is recorded as a case of congenital malformation. Very rarely is the contour of the gray matter in an artificial duplication of the cord preserved as perfectly as in these sections. (It is highly probable that Fürstner and Zacher have neglected to picture or record completely the deflections of the white matter in the doubled region in their case.)

In Fig. 64 are shown two symmetrical areas of rarefaction of the gray matter which border on the production of cavities.

Figs. 67 to 69 are taken from a set of serial sections through the upper swollen region in Fig. 23, and are good examples of the general features of the ordinary results of duplication of the cord from bruising. If a bruise of the cord does produce a duplication, nine times out of ten the sections have the irregular contours of the gray matter in these figures rather than the more perfect and exceptional results shown in the two preceding figures.

These sections show that two portions of the cord have been telescoped together, so that their anterior surfaces

face each other. One of the portions (the upper one in the drawings) corresponds to the first dorsal segment, while the other portion belongs to the lower cervical region.

In Fig. 67 the gray matter in the upper section appears to have the form of two perfect segments of gray substance, but the left-hand segment, although having the shape of an anterior horn, contains no ganglion cells, and is really only a portion of the gray commissure and posterior horn which has been artfully deformed so as to appear like an anterior horn. In Fig. 68 the real companion anterior horn makes its appearance in the upper section, and in Fig. 69 the gray matter, although somewhat deformed, assumes the characteristic topography of the first dorsal segment. It will be seen that while the upper section increases in area the lower cervical section diminishes in extent, so that the two segments have been spliced together as one would slide two wedges together with the apex of each one lying against the base of the other. The artefacts of the white matter are not shown in the drawings.

Figs. 70 to 75. These sections are selected from a series from the lower swollen region in Fig. 23. This series is exceedingly interesting in demonstrating the complex and irregular character of artificial duplications of the cord.

In Fig. 70 a portion of the dorsal cord lies in front of and is partially fused with one of the lower cervical segments. In the dorsal cord the right anterior horn, *a*, has been dislocated from the gray commissure and adjacent gray matter. In Fig. 71 the same dislocated horn, *a*, together with its surrounding white matter, has been thrust out laterally on the left side into a tongue-like projection. The dislocated horn looks as if it were stretched out into a long, slender mass; but this is not the case. The seemingly extended portion of this dorsal is really contributed by the right-hand (in the drawing) cervical horn, which is almost connected with the dorsal horn, *a*, by a curved spur-like projection.

In Fig. 72 the source of the increased volume of the dislocated horn, *a*, is easily understood. The substance of the right-hand cervical horn has flowed out into a long, slender mass and joins the dislocated dorsal horn. Fig. 72 is the only one of the series in which the changes in the

white matter have been shown. Fig. 73 shows very much the same conditions as in 72, but a new feature in this section is the fragment of gray matter, *b*. Besides sending off laterally and obliquely upward the long, slender column fused with the dislocated dorsal horn, *a*, the right cervical horn also expels a portion of its substance into the dorsal cord designated by *b*. In sections 72 and 71 the connection between the cervical horn and its appertaining fragment, *b*, is lost, and in 72, and especially in 71, the fragment *b* appears to belong in the dorsal cord as a dorsal horn, whereas it really belongs to the cervical gray matter. Thus, by the expulsion of the cervical gray matter, and by their fusion with the gray matter of the dorsal cord in two different places, the topography of the sections becomes very complex.

Fig. 74 should be reversed from right to left. The dislocated dorsal horn is still present at *a*, while the bridge of gray matter connecting it with the cervical horn has disappeared. Most of the ganglion cells of the cervical horn in question are absent. They are to be found in the higher levels in the fragment *b* and in the gray portion fused with the dislocated dorsal horn in Figs. 73, 72, and 71. In Fig. 74 the gray commissure has also united with the adjacent cervical horn.

In Fig. 75, the final member of the series, the appearances are very curious. A dorsal horn and a cervical horn of the same side have disappeared from the section and the remaining dorsal and cervical horn of the opposite side are joined together by a slender band of gray matter. The gray commissures of the two united horns are likewise joined together. The missing dorsal horn corresponds to *a* in the different sections, and the absent cervical horn has been expended in furnishing the fragment *b* and the elongated column fused with *a*.

It must be confessed that the appearances of such a section as 75 are very deceptive, and would be quite inexplicable without a set of serial sections, and would tempt an observer to think of a congenital malformation.*

* The material was hardened carefully in the usual way in Müller's fluid, and subsequently in alcohol. The celloidin-imbedded sections—cut in serial sets through the bruised regions in most of the cases—were

IV. The Minute Structural Artefacts due to bruising the Cord.

Thus far in these notes only the topographical changes in the cord incident to bruising have been considered. Besides these topographical changes, bruises of the cord also produce another important set of *structural* changes in the elements of the cord substances. These minute structural artefacts from bruising will be briefly alluded to now, while their more detailed description will be reserved for a subsequent paper, for these artefacts are liable to be misunderstood or mistaken for the results of pathological processes.

These minute structural artefacts occur in both the gray and in the white matter.

In the *gray matter* the principal structural artefact is the *peculiar rarefaction* or porous condition of the gray horns which has already been sufficiently described in Figs. 5, 58 to 61, and 64 (see also Drummond's case in Section VI). There are also occasionally various mechanical changes in the *ganglion cells* in bruised regions of the cord.

In the *white matter* there are minute changes in the *nerve fibers* and *neuroglia*. The nerve fibers show mechanical disintegration of the myelin, alterations in their caliber and in the thickness of the axis cylinders. A group of nerve fibers, while maintaining their proper direction, may be compressed together so as to stain differently and appear degenerated to the naked eye or under a low power. In fact, these mechanical changes in the nerve fibers have been mistaken for lesions of the cord. (See Feist's first case, Section VI.)

Bands of neuroglia may be squeezed together mechanically, and take on a very deceptive appearance suggestive of neurogliar thickenings from disease. Portions of the superficial neurogliar zone may be thrust inside of the cord and form suspicious little islands near the periphery, which might call to mind syphilis or some other chronic inflam-

stained with Weigert's hæmatoxylon method, but principally by the picro-acid-fuchsin method (described in Laboratory Notes of Technical Methods for the Nervous System by the writer in the *New York Medical Journal* for July 20, 1889). The drawings were all made from individual sections with the camera lucida, and are magnified from five to fifteen diameters.

matory process. Bits of the peripheral extremity of the posterior horn may be rarefied and spread out into the midst of the neighboring white matter in such a way as to give an entirely different appearance from the usual compact look of the horn in its proper place, and present such a confusing picture that the foreign mass may not be recognized as tissue of the posterior horn. The associated artefacts, whether topographical or minute, ought to correct any wrong impressions about such a condition of the posterior horn or neuroglia.

The occurrence of *cavities*, *clefts*, *splits*, and *hæmorrhages* from bruises of the cord has already been alluded to. Artificial hæmorrhages may fill up the central and the perivascular spaces surrounding the sulco-commissural vessels. Bruises may also produce collections of fluid and œdematous regions in the cord substances.

It may be suggested incidentally that it might be of importance to have in mind the possible artificial origin of certain hæmorrhages in the central nervous system in various medico-legal cases that might arise.

When a diseased cord is bruised and the minute structural artefacts become mingled with or modify diseased areas in the sections, it often becomes an exceedingly difficult matter to recognize or understand the lesions, and to determine how much of the changes are due to disease and how much to artificial changes.

In a normal cord these minute structural artefacts are easy to recognize from their somewhat characteristic features, from the absence of disease process in their vicinity, and from their usual association with topographical artefacts of the gray and white matters. It is well to note, however, that under certain circumstances a bruise may produce minute changes in the cord substances, and yet not be severe enough to change their topography.

General Remarks.

Thus it will be seen in this section that minor topographical artefacts, complex duplications of the cord, and deceptive minute changes may be produced in the cord substances—may be produced at any time at the autopsy by careless technique or bruising.

In the next section we shall find that just such artefacts are described as congenital malformations or as the results of pathological processes, and that these artefacts are also rather largely and attractively speculated upon in their supposed relations to the production of disease processes.

EXPLANATIONS OF THE PLATES.

PLATE I.—Sections from a case bruised unawares at the autopsy. The case presented obscure nervous symptoms, and the artificial changes were noted in the autopsy protocol without being attributed to bruising. Figs. 1 and 2 show how the gray segments have been split open by deflected bands of white matter the fibers of which pass horizontally in the section, as shown by the wavy lines in the section. Figs. 3 and 4 show rather extensive artificial deformities of the gray matter, with extra portions of the same driven up from other levels, and various columns of deflected white fibers. Figs. 5 and 6 show the appearances of sections from the surfaces of segments, where in the fresh state after incisions the cord substances have welled up out of the interior.

PLATE II.—Sections from various cords, unintentionally bruised at the autopsy, found in the laboratory collection of spinal cords. In Fig. 7 part of the left posterior horn, with the column of Clarke, has been thrust out into the anterior fissure. In Fig. 10 the anterior horns have been fused together in a common horseshoe-shaped mass; *x x*, columns of Clarke; *c*, central canal. Fig. 12 shows how a limited column of deflected white fibers have indented the anterior horn.

PLATE III.—Sections from the cord in a case of chorea, bruised unawares at the autopsy. There were no external gross changes in the cord at all from the bruise. In Fig. 13 there is a deficiency in the gray matter of the posterior horns, which appears in the serial sections above, in Figs. 14 and 15, and apparently extra portions of gray matter; the horizontally dispersed nerve fibers in the white matter show very well in all of these sections. In Fig. 15 the mechanically dispersed nerve fibers surround the dislocated gray fragment in concentric lines.

PLATE VII.—Gross appearances of bruises of the spinal cord. Drawn from hardened specimens. Figs. 21 and 22 show extrusions of the cord substances which look very much like tumors. In Fig. 21 the cord was bruised unawares at the autopsy. In the remaining specimens the bruising was done intentionally. In Fig. 23 the cord has been very severely bruised, so as to telescope different segments together, producing duplication of the organ. A number of small extrusions of

the cord substances have produced multiple wartlike excrescences on the surface of the cord. Fig. 24 shows the transit of a displaced plug of white matter from one region in the cord to another level.

Plate VIII.—Sections from an experimental bruise of the spinal cord in the lower cervical region. Figs. 25 to 30 are taken from a serial set of sections from the lower cervical region, and show a progressive disfiguration of the right anterior horn, together with the characteristic deflections of masses of nerve fibers in the white matter. An extra or dislocated fragment of gray matter appears in Figs. 29 and 30. The wavy lines in these figures, as well as in all of the succeeding plates, indicate the deflected bundles of white matter passing horizontally or obliquely through the sections.

Plate IX. — Microscopical appearances of experimental bruises of the spinal cord. Fig. 31 is the last member of the preceding series. Fig. 32 shows how nearly all of the left gray segment has been shifted to another left, while the right gray horns are not at all damaged. In Fig. 33 the left horns are so uniformly compressed that hemiatrophy is suggested. Fig. 34 shows separation of portions of the anterior horns by deflected bands of white fibers. Fig. 35 shows a small bit of dislocated gray matter from another level lying between the asymmetrical gray horns. The white matter in all the sections shows the same changes as in the preceding plate.

Plate X. — Microscopical appearances of experimental bruises of the spinal cord (continued). In Fig. 37 the anterior horns and gray commissure are absent, and some of this missing gray matter appears in Fig. 39, giving the appearance of a third anterior horn between the two normally situated anterior horns. The sections are selected from a serial set, and show especially well the horizontal and other dispersions of the white matter from bruising. In Fig. 39 note the little peripheral nodular masses of bruised fibers which have burst through the periphery of the cord.

Plate XI. — Microscopical appearances of experimental bruises of the spinal cord (continued). In Fig. 40 minute structural artefacts in the nerve fibers in the column of Goll, incident to bruising, simulate a patch of sclerosis. The spur-like projection of gray matter in Fig. 40 is broken up into fragments in Fig. 41. Figs. 43 to 45 show one of the most common and uniform effects of bruising—viz., rupture of the centrally situated cord substances, with dislocation of the gray commissure and separation of the gray segments on either side

Plate XII.—Microscopical appearances of experimental bruises of the spinal cord (continued). In Figs. 46 and 47 (from

serial sections) the source of the extra fragments of gray matter in 46 can be seen in 47, where the gray matter is deficient on the left side. In Figs. 49, 50, and 51 (from serial sections) the absent horn in Fig. 49 appears in Figs. 50 and 51. Fig. 49 should be reversed from right to left.

Plate XIII.—Sections from experimental bruises of the spinal cord (continued). Fig. 52 shows dislocation of the right anterior horn and dispersed white fibers passing obliquely instead of horizontally through the section. Fig. 54 shows how nearly all of the gray matter may be driven to another region, tending to produce more or less of a duplication of the cord in the new level. In Fig. 55 the whole of the white matter is occupied by deflected nerve fibers. Fig. 56 belongs among the artificial duplications of the cord in the next plates.

Plate XIV.—Artificial duplication of the spinal cord resulting from a bruise. Figs. 57 to 61 are from a serial set of sections through a bruise of the cord, in which a segment from the dorsal region has been dislodged and telescoped down through the anterior fissure (see Fig. 58) to another level in the cord. The displaced segment has a pair of anterior horns which eventually (in Fig. 61) become fused two by two (Fig. 61, *a*, *b*) with the anterior horns of the cord at the new level.

Plate XV.—Sections from several forms of artificial duplications of the spinal cord produced by bruises. Figs. 62 (see Fig. 17) and 63 (see Fig. 18) show extrusions of the white matter resulting from an attempt to produce doubling of the cord. *The dark spots in the right anterior horn in Fig. 62 indicate hæmorrhages produced by the bruising.* Fig. 66 is from a section from the cord in Fig. 22. Figs. 64 and 65 (from the cord in Fig. 19) show an exceptionally perfect form of artificial duplication. Figs. 67 to 69 show a telescoping together of the first dorsal and lowermost cervical segments. Changes in white matter in Figs. 64 to 69 are not indicated.

Plate XVI.—Artificial duplication of the spinal cord due to bruising. The gross appearances are shown in the lower swollen region in Fig. 23. Portions of the dorsal and cervical cords have been fused together. One of the dorsal horns has been dislocated and thrust out laterally at *a*. The right-hand cervical horn sends out a long, slender portion of gray matter, which becomes continuous with *a*. The fragment *b* also belongs to the right cervical horn (Fig. 73). In Fig. 75 a dorsal and a cervical horn of the same side have disappeared, and the remaining cervical and dorsal horns and their gray commissures are joined together.

Plate XVII.—Seguin's case. Reported by Delafield. Two consecutive spinal segments have been telescoped together by a

bruise of the cord. The twelth dorsal segment, with its deformed gray segments and dispersed white matter, has been thrust down over the posterior surface of the first lumbar segment, which is but comparatively little deformed except in the lengthening out of the posterior horns in Fig. 2. The two areas of descending degeneration in the crossed pyramidal tract, in the lumbar segment, can not be recognized in the dorsal segment, because of the mutilation of the white matter. (The columns of Clarke in the transported segment are represented by the darkly shaded areas in the gray matter.)

Section VI.

AN ANALYSIS OF THE ERRONEOUS CASES OF SPINAL-CORD MALFORMATIONS PRODUCED BY MANIPULATION, OR DISEASE, OR BOTH COMBINED.

Of the thirty-two cases in the literature of spinal-cord malformations, the only real instances of true heterotopia or malformation of which we feel positive are the eight cases described in Section II; these are the only cases on record where the extra portions of gray matter (Pick) or the misplaced nervous substances (Pick, Cramer, Kronthal, and Virchow) are actually of developmental origin.

The supposed malformations in the remaining twenty-four cases analyzed in this section are either simply cases of bruising or, in a few instances, the results of disease. In fifteen of these cases (cases No. 2, 4, 5, 6, 7, 9, 10, 11, 13, 14, 16, 18, and 20) the supposed malformations were produced by bruising alone. In five of the cases (3 (?), 8, 12, 15, and 17) the alleged malformations were not entirely caused by bruises, but were due to some extent to destructive and deforming diseases. In these five cases the cord had been damaged during life by acute myelitis, and the bruising or handling at the autopsy had increased the distortions of the gray and white matters induced by the myelitis. Acute myelitis renders the cord more susceptible to bruises, and the resulting deformities are always more extensive than in a normal cord. In four of the cases (3, 4, 8, and 14) artificial duplications of the cord, due to bruising, were mistaken for congenital malformations. Two of these cases (3 and 4) show a very perfect form of artificial duplication, while the remaining two (8 and 14) show less perfect or incomplete forms of duplication artifacts. In

one of the cases (No. 1) the so-called malformation appears to have been produced by tabes dorsalis.

The mere fact that there were no symptoms or bodily defects in any of these cases pointing toward the cord deformities should throw a great deal of doubt upon their supposed preformed origin; yet the cases have been quite unreservedly described as malformations existing during life. In order to make the review of these perfectly clear and brief—and especially for comparison with the drawings belonging to Sections II and IV—we have reproduced the original plates accompanying the articles analyzed in this section.

No. 1. *Case of Kahler and Pick* (22), *1879.*—The writer has hesitated about classing this case among the erroneous instances of spinal-cord malformations, but it has been included with them, because it seems difficult to exclude disease—tabes dorsalis—as the factor producing the changes described by the authors as a malformation.

The malformation in this case consisted in an abnormal narrowness or thinning of the posterior horns, so that the contour of the gray horns resembled the type seen in animals, such as the ungulate or carnivorous orders, rather than the classical type characteristic of the human cord. (See

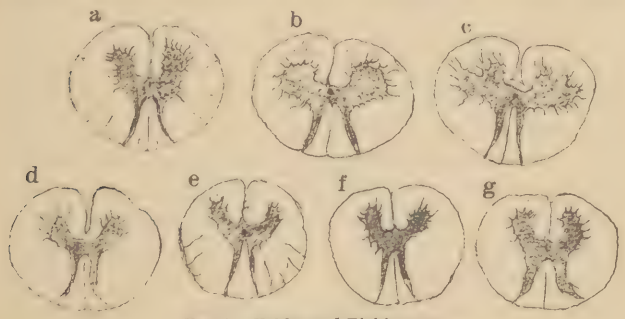

FIG. 1.—Kahler and Pick's case.

Fig. 1.) The posterior or white columns were also very narrow, and the columns of Clarke appeared to be defective in the middle dorsal region, although normal in the lowest dorsal and uppermost lumbar regions.

The cord came from a case of tabes, and showed the usual lesion in the posterior columns. The authors, how-

ever, dismiss the question of the production of the change in the posterior horns by the tabetic lesion in the posterior white columns, because these were not retracted at their peripheral margins, and the tissues of the narrowed posterior horns did not show the structure of the old and shrinking stage of sclerosis. But these are not very convincing reasons for rejecting the tabetic origin of the deformity.

After dismissing the tabetic origin of the deformity, the view is presented that the change in the shape of the posterior horns was a congenital malformation due to a deficient embryonal site (*Anlage*) or development of the posterior columns or horns. The suggestion is finally made that the primary starting point in this defective development of the cord was localized in a deficient volumetric fœtal arrangement or subsequent development of the *bandelettes externes* or columns of Burdach.

The shrunken condition of the columns of Clarke leads toward this suggestion, because of the anatomical relation, which the writers note, between the bandelettes and the columns of Clarke. The whole context intimates that the primary defective growth of *bandelettes externes* would operate in a secondary manner to diminish the columns of Clarke and the posterior horns. A question is presented as to whether the nature of the developmental error in the bandelettes consists in an arrested or faulty development of the medullary sheaths of their nerve fibers.

In consequence of their view of the developmental origin of the supposed malformation, the authors suggest the hypothesis that the malformation of the cord tended to invite the occurrence of the tabetic lesion, and that the neurotipathic disposion in general sometimes depends upon defective development.

This is the first instance in the literature on heterotopia in which the supposed causal relation to disease of these alleged spinal malformations, or the dependence of the neuropathic disposition in general upon defective development, is discussed, and it is for the reason that this discussion in Kahler and Pick's paper, and in the next article of Fürstner and Zacher, has served as a text for the elaboration of the same theme by subsequent writers, that the

matter of dependence of disease and neuropathic disposition in these two misunderstood deformities is reviewed with some detail.

As to the case of Kahler and Pick, notwithstanding their high authority, it seems to me that the case is simply one of atrophy of the normally developed posterior horn, produced directly or indirectly by the posterior sclerosis, such as is observed occasionally in cases of long duration. The remarks of the authors, based on their own case, concerning the neuropathic disposition, do not seem to be justified by the facts.

The writer has met with one instance of tabes of very long standing—an old orderly at Bellevue Hospital, who had tabes for twenty-five or thirty years—in which the long-continued posterior sclerosis had produced changes in the posterior horns quite similar to those in Kahler and Pick's case. In the writer's case the posterior sclerosis involved the whole cord up to the nuclei of the posterior columns, which were hardly at all retracted at their peripheral margins, and although it was not easy to decide whether the sclerosis directly involved the posterior horns, these were atrophied in the upper lumbar, dorsal, and, to a less striking extent, in the lower cervical regions, so that the sections resemble *a*, *d*, *e*, *f*, and *g*, in Kahler and Pick's case, Fig. 1 (*a*, upper cervical; *d*, upper dorsal; *e*, *f*, mid-dorsal; *g*, upper lumbar). With the exception of *b* and *c*, the shrinking of the posterior columns and atrophy of the posterior horns shown in the other figures are not remarkable in an old case of tabes. In *b* and *c* the deformity produced by the tabetic lesion is extraordinary, and much more pronounced than in the writer's case.

Kahler and Pick's case was examined before the discovery of the important staining methods of the present time, and before Lissauer (Fortschritte der Medicin, 1886) called attention to the secondary atrophic condition of Clarke's columns, which the writers do not apparently appreciate, and which we have since found to be quite a uniform and characteristic feature of tabes.

Taken altogether, the deformity seems quite conclusively to be secondary to the tabetic lesion, and not at all responsible for its invasion of the cord.

No. 2. *Schultze's Cases* (23), *1881.*—Schultze, in an address on the relation of developmental anomalies to the neuropathic disposition, notes that it not infrequently happens that peculiar and but little known abnormities of the cord structure, particularly in the distribution of the gray and white matter, occur in cords subject to chronic lesions. By this, Schultze is referring to the condition of heterotopia and instances two cases.

First Case.—In the first case, one of amyotrophic lateral sclerosis, *the gray matter of one side of the lumbar enlargement was pierced or traversed centrally by a band of white matter* which was not degenerated. In the cervical part the superficial neuroglia did not go around over the anterior median white columns into the anterior fissure, but passed directly through the middle of the anterior horn.

Second Case. — In the second case, one of general paresis, in the dorsal and upper lumbar regions *there were curious distortions of the configuration of the gray matter, so irregular as to be difficult to describe. Portions of the gray matter were dislocated and isolated from the parental masses by interfering bands of white matter.*

There are no drawings of the appearances in these cases, yet, I think, they can be safely pronounced as due to artificial means or bruising; for the deformities do not correspond at all with the characteristics of the true cases of heterotopia in Section II, but are counterparts of the appearances shown to be due to bruises in Sections IV and V. The "interfering bands of white matter" and the "dislocated portions of gray matter" which Schultze speaks of betray the artificial origin of the deformity, and these are simply analogous to the bruised and deflected cord substances abundantly shown in our drawings.

It is unfortunate that Schultze lent the authority of his name in impressing these two cases of artifacts of the cord into the service of the neuropathic disposition, and advised the collection of statistical material of like cases to throw light on their relation to the induction of spinal diseases; for his example has been industriously followed, and hardly a single subsequent writer, mistaking bruises for malformations, resists the belief that the supposed malformation may

have had some causal relation to the disease for which the cord was removed.

In this way the relation of their misunderstood cases to the neuropathic disposition, indicated by Kahler and Pick, Fürstner and Zacher, and especially Schultze, has been followed from one writer to another, until to-day, reasoning by analogy from the recognized tendency which other congenitally malformed organs have to invite disease, it is made to appear that the spinal cord is also liable to become diseased when affected with these supposedly frequent malformations which in most instances are nothing but autopsy bruises.

No. 3. *Case of Fürstner and Zacher* (24), *1882.*—This case is described rather diffusely and is difficult to review concisely. It will be sufficient to show that the case is one of bruising of the cord rather than to be concerned with the deductions, which appear all through the paper, based upon the error of the writers in mistaking an autopsy bruise for a congenital malformation. Congenital malformations of both the cord and brain are described.

The alleged cord deformity was of the heterotopic order and also consisted in a doubling of the cord, but I have no hesitation in pronouncing the deformities to be due to an autopsy bruise—a bruise which, in addition to the production of the ordinary minor displacements and asymmetries of the cord substances, was violent enough, at one place, to telescope one portion of the cord down over another so as to form doubling of the cord over a limited space.

The cerebral changes consisted in an atrophy of the left frontal lobe, where there was a sac of fluid distending the pia mater, and a widening of the lips of the Sylvian fossa, all of which the writers are inclined to consider a congenital malformation — apparently considerably influenced in this conclusion by the existence of the cord malformation. These cerebral changes do not positively indicate a congenital origin; they might have been well enough the effects of disease of the Sylvian vessels, which element the writers do not at all eliminate, nowhere mentioning the condition of these vessels. But they think that the two sets of deformities of the brain and cord were more or less

naturally associated with each other in indicating some weakness of development of the whole central nervous system.

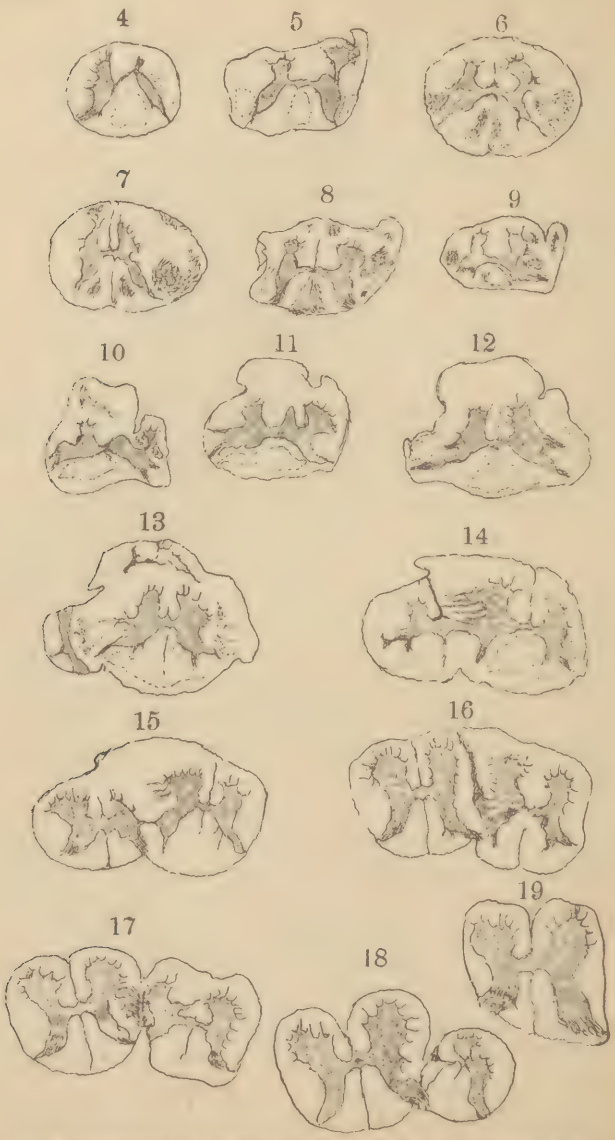

Fig. 2.—Fürstner and Zacher's case. Artificial duplication of the cord.

The introduction of the paper is taken up with a general discussion of the induction of the neuropathic disposition by such deformities of the cord, and presents the view that the case itself is a specific example of how the weak anatomical constitution of the nervous system made it prone to disease (there were dementia paralytica and myelitis). An outline of the clinical history is given here to show that there were no symptoms whatsoever of the supposed cord malformation, which in itself militates strongly against the idea that any such extensive distortions as in this case could have existed during life. The autopsy report of the cord is reproduced in full, for it is a good description of severe bruising of the cord. The report of the microscopical examination is very long, and only enough of it is abstracted to show that the appearances of the sections are counterparts of the artifacts already described in the two preceding sections.

Clinical History.—A left-handed man, aged fifty, *had no bodily deformities*, and had worked in a caisson for some time. He had dementia paralytica with disturbances of equilibrium and rigidity; there were also tremors and sensory disturbances. The patellar reflex was absent, and later a myelitis produced paraplegia, and finally death.

Autopsy.—The spinal column was not deformed and had a normally developed single dura mater and arachnoid coat lining it. After opening the spinal canal, the cord was observed to have a peculiar shape. In the lowermost dorsal region it was exceedingly narrow, but in the lowest dorsal and upper lumbar regions, for a distance of four to five centimetres, the cord was swollen and irregularly thickened, so that a condition resembling an intramedullary tumor was produced. The surface of the thickened portion was very uneven and showed numerous indentations, furrows, and prominences. Altogether this thickened portion of the cord appeared as if the real cord were surrounded by a broad zone of extra nervous material upon which the pia mater lay. *The cord just above the swollen region was so soft that it flowed out for a short distance, and was lost in spite of the efforts made to keep it intact.*

Microscopical Examination.—The spinal cord showed two sets of alterations—malformations and pathological

changes. The cord was free from abnormities to the seventh or eighth dorsal segments, where a series of changes in the configuration of the gray matter began and developed more extensively as the sections passed downward.

At the uppermost level in the cord the deformities make their appearance in this manner: The right anterior horn becomes smaller than the left, due to the horizontal passage of a bundle of white fibers from the right to the left side of the cord, which gradually becomes larger and larger, and replaces entirely the right anterior horn, and then spreads out like a brush at the periphery of the cord. (See Fig. 2, *4*.)

The further course of this anomaly, beginning as shown in *4*, could not be traced accurately in the lower dorsal region, for this region of the cord, as already mentioned in the autopsy report, flowed out in handling the cord and was not hardened in its proper position. The extruded portion was hardened, however, and, by comparing the configuration of the sections with *4*, the authors think it probable that the continuance of the deformities is in the order of the figures from *5* to *11*.

Fig. 2, *5*, probably corresponds to the ninth or tenth dorsal segments; the white matter in this section was arranged in various bundles, running in irregular directions, and the white commissure was absent. (The figures have been drawn on such a small scale in the original article that they are of hardly any value at all in showing the arrangement of the white matter.)

Fig. 2, *6* and *7*, from the extruded portion of the cord, are nearly normal, sections 8 and 9, from the lowest level of the same portion of the cord, show an asymmetry of the gray matter and the same sort of bundles of white fibers running about in abnormal courses and directions as shown in *4* and *5*.

The remainder of the sections illustrate the appearances in the swollen voluminous portion of the cord described in the autopsy notes. Sections 10, 11, and 12 show well enough what has happened in this case, and the lengthy description in the original paper may be omitted. The most striking feature about these three sections is the accession of an extra amount of abnormally arranged white matter in

the anterior half of the cord in front of the anterior horns. The posterior horns are also spread apart and flattened out.

In section 13, besides the extra amount of white matter in the anterior portion of the cord, there is also an extra volume of white matter on either side of the cord. On the left side, the extra portion of white matter incloses an accessory fragment of gray matter, which has the arrangement of the gray commissures. The white fibers toward the periphery of this section are arranged irregularly (horizontally ?), but much of the remaining white matter was vertically arranged.

The succeeding five sections show the doubling of the cord in the swollen portion, and in *19*, below this swollen portion, the cord structures are normally arranged.

Remarks.—It is unnecessary to discuss the minutiæ of microscopical appearances in this case to come to the conclusion that they are artifacts. The autopsy notes of gross appearances of the cord indicate so plainly that the cord had been severely bruised in its removal that it is surprising that the authors should have mistaken these appearances for congenital malformations. The writers plainly describe the reduced size of the cord in the lower dorsal region, and a deficiency of the cord at the lowermost dorsal portion ; yet it does not occur to them that this deficiency was partly caused by the telescoping downward of a segment of the lowermost dorsal region over the lumbar cord, which produced the swollen and voluminous appearance grossly. The sections also show this to be the case. In sections 16, 17, and 18 the shape of the gray horns of the supposed accessory cord on the right side shows that it has been pushed downward from a higher level alongside of the primary cord. The right-hand cord segment in these figures has the appearance of the twelfth dorsal or first lumbar segments, while the left-hand segment, over which the former has been telescoped, corresponds to the third or fourth lumbar segments ; so that some of the portion of the cord which the authors supposed to be deficient is the right-hand segment in sections 16, 17, and 18.

The artificial doubling of the cord in this case is remarkably perfect. I have, however, succeeded in one of many experimental attempts in duplicating such appear-

ances. There has always been in these experiments a good deal of distortion of the gray and white matters in both the cord itself and in the telescoped segment. It is very probable that there were deflections of the white matter in these doubled sections of Fürstner and Zacher not indicated in the drawings, which are all on too small a scale to give any satisfactory idea of the artifacts of the white matter.

When we sum up the microscopical features of the other portions of the cord, we find that there are contortions and dislocations of the gray matter, and diverted bundles of white matter running in abnormal directions. These, especially the bands of white matter running collectively or dispersed in a horizontal plane, are characteristic of careless manipulation.

Portions of the text embracing the microscopical examination are not clear as to whether the lesions described were actually due to pathological processes, or whether they were simply due to the minute structural artifacts in the nerve fibers of the crushed areas in the white matter inflicted in the bruising, which we have shown in Section V may simulate very closely some of the lesions of the cord.

Section 4 of this case should be compared with a similar appearance among the experimental bruises shown in Fig. 48, Plate XII.

At the conclusion of the paper the writers briefly allude to a second case of cord deformities of this kind.

4. *Seguin's Case* (25), *1872.**—This case seems to have been regarded more as a new growth or "myelinic tumor of the cord" than a malformation. The following account of the case is taken from Delafield:

"The patient was a woman thirty years of age. Two years before her death she received a severe blow on the back of the head. After this, first one side, then all of the limbs became paralyzed. From this condition she recovered suffi-

* A description of this case, with drawings, was sent to the *Archives de physiologie* by Professor Seguin in 1872; these were lost by the editors, and the case was not published until Professor Delafield made a note of it in *Post-mortem Examinations and Morbid Anatomy*, New York, 1872, p. 65; and in *Handbook of Pathological Anatomy*, New York, second edition, by Delafield and Prudden. During the past year Dr. Seguin has kindly sent me sections and additional clinical notes for a renewed study of the case.

ciently to walk; but, after a short time, she again grew worse, and both arms and legs were paralyzed and contractured. She died of bronchitis. At the autopsy there were found two tumors on the spinal cord within the dura mater.

"The upper tumor was on the anterior face of the cord, just below the decussation of the medulla, and of the size of a pigeon's egg. It was composed of branched connective-tissue cells with many round cells.

"The lower tumor was on the posterior face of the cord at the upper part of the lumbar enlargement. It was of the size and shape of a large flattened cherry. It was composed of portions of two spinal cords fused together, with their long axes parallel to that of the normal cord. There were four patches of gray matter, having the shape of the cornua, containing ganglion cells, and joined two and two by regular commissures, in each of which there was a central canal. These gray portions were surrounded by tissues resembling the white substance of the cord."

Remarks.—There were absolutely no symptoms referable to this lowermost tumor, and, from a study of the sections (see Plate XVII, Section V), I can unreservedly pronounce this appearance to be the results of bruising at the autopsy, although no detailed notes about the cord removal are to be had. The duplication, as in the previous case, is remarkably perfect, and is probably exceedingly seldom produced in routine autopsy work.

In Plate XVII, Section V, it can be seen, from the shape of the gray horns in the duplicated portion, that the latter have come down from a higher level in the cord. The duplicating portion corresponds to the twelfth dorsal segment, while the cord itself is from the second lumbar level. In the telescoped segment the white matter is disarranged and crushed, so that the two areas of descending degeneration in the lateral columns, from the cervical lesion, can not be seen. The posterior horns in the transported portion are absent. There is also a large split in the center of the section due to an imperfect apposition of the parts at the time of the blow, or a subsequent shrinking of the softened transported segment. In Fig. 2 (Plate XVII, Section V) there are some fragments of detritus of the white matter clinging to the walls of the split.

From the description, the gross appearances of this cord may have resembled Fig. 18 or 19, Plate VI, Section V.

The cases of Seguin and of Fürstner and Zacher have two points in common, which can be explained very easily by the bruising. In the first place, the two portions of the cord in the duplicated region are from consecutive levels. In Seguin's case the lower part of the last dorsal and the upper part of the first lumbar segments have been telescoped down over the next lower segment—the second lumbar. In Fürstner and Zacher's case these same upper segments and, in addition, perhaps, an upper bit of the second lumbar segment, has been thrust down over the next lower segments—the third and fourth lumbar. Although we could not deny that such a striking sequence of segments might occur in a hypothetical case of doubling of the cord of real congenital origin (there are no genuine cases recorded outside of double monsters), yet we should hardly expect so uniform a sequence of segments in genuine cases of doubling, if they exist at all in adults, as is almost invariably the rule in bruises.

In the second place, both cases of doubling occur at approximately the same levels in the cord—the dorso-lumbar junction. The explanation of this coincidence, I believe, is that the cord becomes so much more difficult to remove in precisely this region from the depth and strength of the vertebral arches, that it is hard to accomplish much with the saw, and the chisel, shears, and mallet are used instead. So in this way bruises are more frequent and most extensive in the dorso-lumbar region where the cord is hardest to remove, and among the many bruises inflicted in this region, these two have produced a doubling of the cord.

A striking thing about Seguin's case is that the telescoped portion did not leave any deficiency from whence it came sufficient to have been noted when the cord was exposed. But, as noted before in Section V, the deficiency corresponding to a doubled portion of the cord is often not at all striking to the eye in the fresh condition, for apparently the membranes over the deficient part do not always collapse, so that the cord, in some cases of doubling, may not show any striking deficiency, above or below the doubled region.

No. 5. *Drummond's Case* (26), *1881.*—This case, together with the following one of Bramwell's, is interesting, from the implied suspicion with which the deformities were regarded in relation to pseudo-hypertrophic paralysis before the entirely peripheral nature of this disease was understood. Both of these cords happened to be bruised in being removed from subjects of that disease. Drummond very carefully refrains from commenting on any relation between the deformity and the disease.

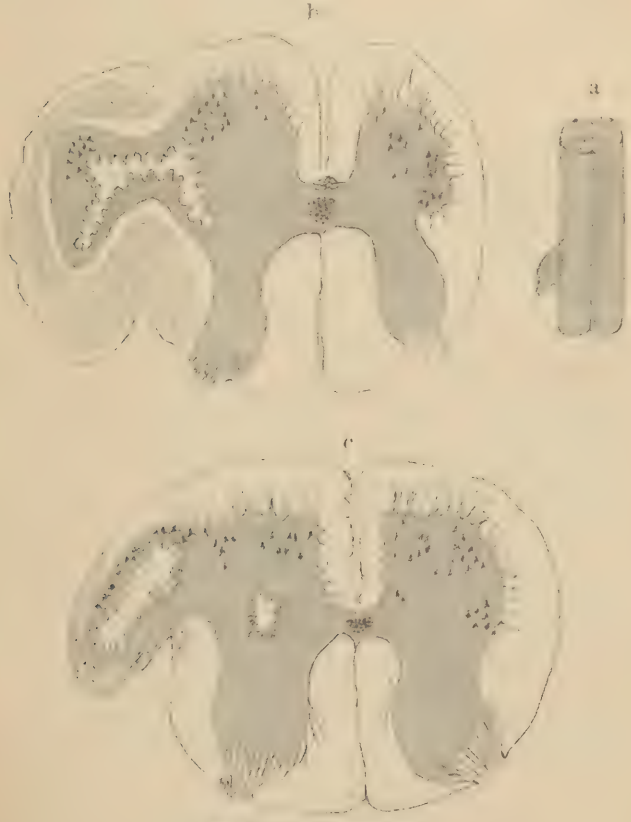

Fig. 3.—Drummond's case.

The cord was removed from a boy, fourteen years old, who had typical pseudo-hypertrophic paralysis.

The cord had a "swelling or tumor," which bulged out

from the anterior part of the left lateral column in the upper lumbar region, which measured nearly a centimetre and a half in length (Fig. 3, *a*). Above, it appeared as a swelling of the ordinary white substance, but below it was bent downward and made an acute angle with the cord.

In making frozen sections through the projecting region of the cord, it was noticed that it was not solid, but consisted of a shell that inclosed some frozen fluid in the center, which disappeared when the section was transferred to water (*b* and *c*). Thus the tumor was cyst-like, having a cavity and a wall of both gray and white matter. While there was disintegration of the gray matter with an accumulation of fluid, *there was no true lining wall of the cyst-like cavity.*

Disintegration of the gray matter of the same nature as shown in Figs. *b* and *c* was also found in the cervical region on the opposite side, but it was not so extensive, and was not associated with any deformity of the cord substances. Drummond's *résumé* of the changes is: "Disintegration in the lateral gray horns, most marked in the lumbar region where the accumulation of serum had caused the cord to bulge out laterally." This observer also very cautiously adds: "I do not mean to assert that this lateral disintegration or tearing was of pathological significance—it might have been due to the manipulation, but I am inclined to think otherwise."

Remarks on Drummond's Case.—Both of the changes in this case—the deformity of the cord substances and the disintegration of the gray matter—are to be regarded as the results of bruising. The absence of any signs of inflammation about the disintegrated areas and the absence of the characteristic structural features of necrosis are not in favor of their having been caused by pathological processes. The whole appearance of the drawings, especially the horizontally deflected nerve fibers in the white matter, indicates that they are mechanical artifacts.

The disintegration of the gray matter described in Drummond's case is the "rarefied" or porous condition which we have frequently shown to be the effect of bruising in the experiments in Section V and in the cases in Section IV. We have found that this rarefied condition of the gray

matter as the result of bruising is more liable to occur with the more extensive deformities, and that it may also occur in bruised cords which are hardly deformed at all as described in the cervical region in Drummond's case. This mechanical disintegration of the gray matter may be of varying grades of intensity. It may appear as a separation of the elements of the gray matter, with a disappearance of more or less of the granular basement substance, so that there is a less dense or rarefied appearance of the gray matter; or the artificial disintegration may be more extensive and form an actual cavity. Figs. 5, 58, 59, 60, 61, 64, Section V, are to be reviewed in connection with Drummond's case on this subject of the production of artificial cavities or rarefied areas in the gray matter.

The finding of this condition in the gray matter in frozen section of the fresh cord in this case is interesting, for it shows that the disintegration is more directly due to the mechanical effects of the bruise than to the subsequent action of the hardening fluids on the bruised regions. We had been led to believe that the lesser grades of disintegration were partly due to the bruise, and partly to the subsequent action of the hardening fluids.

Drummond is the only one of these writers on supposed adult cord malformations who is reserved in his conclusions and appreciates the possible artificial origin of these changes. He nowhere speaks of the changes as malformations, and it is singular that he should imply a preference for the view that the disintegration of the gray matter with the production of fluid was the primary condition and induced the deformity secondarily by pouching out the cord substances beyond the periphery, instead of attributing the deformity to the bruise and the disintegration and production of the ragged-walled cavity to the same cause.

In this case the squeezing of the gray matter in the bruising seems to have pressed out a collection of fluid which appeared in the frozen sections.

No. 6. *Bramwell's Cases, 1885.*—Bramwell* cites two cases of congenital malformations of the cord in his work on the diseases of the spinal cord. The first cord came

* Bramwell. *Diseases of the Spinal Cord*, Fig. 167, p. 334, second edition, 1885.

from a case of pseudo-hypertrophic paralysis, and the second is instanced without any history in the section on congenital malformations of the cord.

The first case showed grossly in the middle of the cervical enlargement that the lateral column of the right side was misshapen by a projection or outgrowth measuring little more than half an inch vertically. The surface of the projecting mass (after having been hardened several weeks

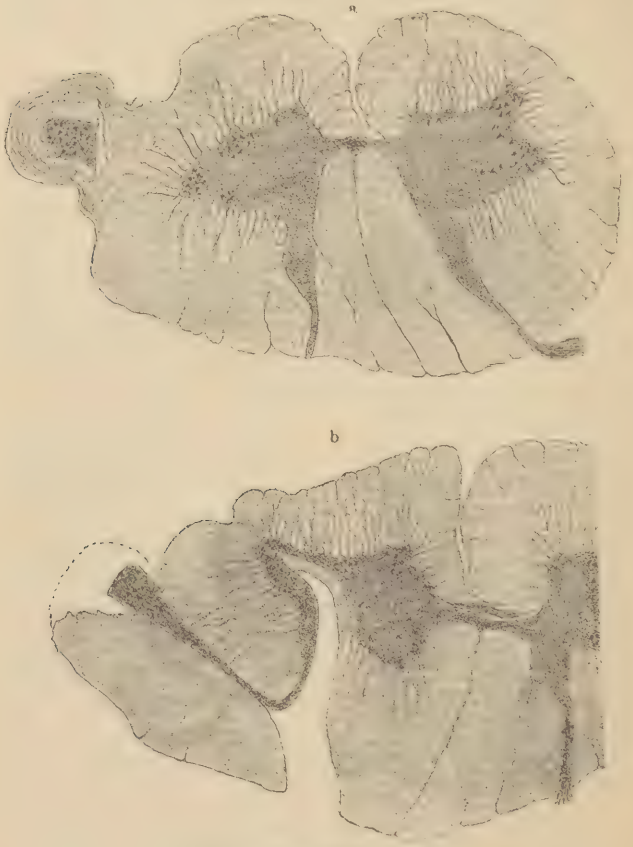

FIG. 4.—Bramwell's first case.

in bichromate solution) was irregular and disintegrated at its most prominent point and exactly resembled nervous tissue in a state of softening. A distinct fissure was also seen

in the lower cervical region, on each side of the cord in the central part of the gray matter.

The microscopical appearances are shown plainly enough in the copies of the author's drawings (Fig. 4) to omit their description. A third drawing from this case (not reproduced here) shows a split in the right posterior horn opening to the free surface of the cord. This split is partly filled up with white matter which looks as if it had been driven into gray matter mechanically. The third drawing also shows small splits or cavities in the opposite anterior horn, of the kind described by Drummond.

Remarks.—There can hardly be any doubt that the deformities in this case of Bramwell were produced by bruising. The description of the gross appearances corresponds to a bruise, and the appearances in *a*, Fig. 4, indicate very plainly a mechanical origin of the deformities. This drawing, as well as the third plate of the same case on page 334 of his book, indicates that the force of the blow had split the cord open, or that some cutting instrument had penetrated the cord—either the chisel, or possibly the scissors used to open the dura mater.

The changes in the white matter, such as deflection and crushing of the fibers which are almost invariably incident to these mechanical deformities, seem to have been somewhat overlooked by Bramwell. In describing Fig. 4, *a*, he says that the extra portion of gray matter had an external layer of white matter, "the nerve tubes being concentrically grouped round the central gray mass and running horizontally and not vertically, as the fibers of the lateral column normally do." This horizontal band of white fibers in *a* is quite characteristic of bruising, and it would be an exception to the rule if there were not also similar changes in the white matter in the section, *b*. If serial sections had been made in the region of *a*, a level would have been found, either above or below, showing a deficiency of the gray matter corresponding to the extra gray mass transported to the level of the drawing by the bruise.

Summing up the case, Bramwell says: "This peculiar alteration in the shape of the gray matter, and the outgrowth from the lateral column, were probably, I think, congenital malformations; whether they had anything to do

with the production of the disease is very doubtful; it is, however, important to remember that a similar outgrowth from the lateral column was present in Dr. Drummond's case; it is therefore probable that in pseudo-hypertrophic paralysis there is a tendency to congenital malformations in the arrangement of the gray matter and the shape of the cord."

The second case, Fig. 5 (page 74 in Bramwell's book), which is simply illustrated in the drawing as a congenital

Fig. 5.—Bramwell's second case.

malformation, but is not described in the text, I believe to be a bruise also. Changes in the white matter from bruising, although not indicated in the drawing, were probably present. In the title to the plate the anterior fissure is said to have been absent. (The fissure was probably displaced or obliterated by the bruise.)

No. 7. *Schiefferdecker's Cases* (27), *1887.*—Schiefferdecker found considerable asymmetry of the gray matter both as to form and position in otherwise perfectly normal spinal cords, and this condition had given no symptoms during life. The first instance was in the cord of a dog,

Fig. 6, *a*, *b*. Both of these sections show a change of form in the posterior horns, and an irregularity in the volume of the right and left anterior horns. The two halves of the cord were also slightly unequal. Above and below this level the cord was perfectly normal and symmetrical.

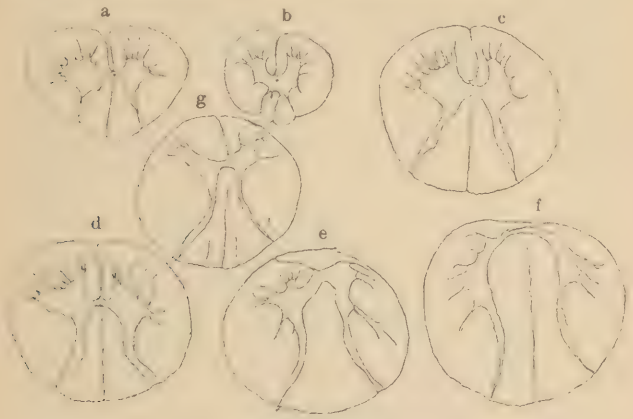

Fig. 6.—Schiefferdecker's cases.

The second case was that of a patient in an insane asylum who had no symptoms of any cord disease, nor symptoms which could be ascribed to the anomaly. The cord membranes were slightly thickened. Fig. 6, *c*, *d*, *e*, *f*, shows plainly enough what has occurred in the cord to dispense with a description of the changes.

Remarks.—Both of these cases are mechanical artifacts. In the first case the mechanical forces were of a mild degree and produced but slight distortions. I have studied quite similar contortions in the cords of dogs and rabbits produced by mechanical means in removing the cord. In Section IV it was shown that the results of bruising a dog's cord resembled quite closely in one or two places the alterations in this case of Schiefferdecker. Fig. 6, *e* and *f*, in the second case are good examples of that rather common result of moderate cord bruising—viz., the central rupture of the cord substances—of which a number of examples are given in the preceding section. Compare Fig. 6, *e* and *f*, of Schiefferdecker's second case with Figs. 40, 41, 43, 44, 46, and 47, Plates XI and XII, Section V.

Schiefferdecker makes a number of anatomical and

physiological conclusions from these artifacts and notes that deformities of this kind seem to be confined to a limited region of the cord, not making the slightest reservation as to their artificial production.

No. 8. *Kronthal's First Case* (28), *1888.*—Kronthal describes at length distortions in the cord from a man having an ordinary subacute destructive myelitis.

Secondary or artificial distortions of the cord substances are so obviously liable to occur in the softened or semi-fluid cords of acute myelitis that but little space would be needed for reviewing this paper were it not for the fact that, being the first article to describe this class of distortions associated with acute destructive myelitis, it is quoted considerably and has exerted an influence in favor of error on subsequent writers. Three other papers describing the distortions in myelitis as malformations have followed Kronthal's article. By reviewing all of the preceding instances of malformations of the cord—with one or two exceptions due to artifacts—which quite regularly occurred in cases of spinal diseases, Kronthal attempts to show that these alleged malformations rendered the cord prone to disease.

The cord was taken from a man twenty-two years old, a plumber, who at first had symptoms of chronic lead poisoning, and during the last five days of his illness developed symptoms of acute myelitis which extended up to the neck. At the autopsy the spinal cord showed two centimetres below its upper extremity a region a centimetre and a half long which was almost fluid, and a second similar nearly fluid region two centimetres long situated twenty-eight centimetres below the upper extremity. The rest of the cord was also softer than normal. Four centimetres above the lower spot of softening the cord was abnormally voluminous; its circumference was increased and looked as if a tumor were present. The cord was not cut open in the softened places, and these regions were hardened *en masse.*

Kronthal's plates will enable us to dispense with his description of the microscopical features of the contortions, which are described in detail in his paper. Among other things, he believes that there was an absolute increase of the gray matter (some sections contained a hundred ganglion cells, etc.), although sections 3 and 14, Fig. 7, show

plainly enough where some of the extra gray matter has come from. Kronthal also interprets the appearances in sections 10, 11, and 12, Fig. 7, as indicating a rudimentary doubled or extra cord, and compares it to the case of Fürstner and Zacher. He does not speak of the artifacts of the white matter very clearly and neglects to indicate them in the drawings. The lesions of myelitis were also found in different stages in the cord. Kronthal dismisses any possibility of manipulation in the production of the deformities, because the cord was not cut open before hardening, and because of the presence of the assumed rudimentary cord.

After reviewing the other causes of bruises, Kronthal sums up his ideas about the proneness of such cords to disease as follows: "It can now be affirmed, considering the last case on record [his own], that we must speak more definitely than formerly and declare that a spinal cord with heterotopia of the gray matter has a diminished resistance to disease."

I have not thought it necessary in the preceding sections to picture or describe the artificial or secondary deformities associated with myelitis. They occur very frequently, their significance is probably generally understood, and it must be generally known that acute myelitis regularly destroys and disfigures the cord substances and generally softens the cord, so that it is hard to remove or handle it without disturbing the topographical relations of the cord matters still more.

One case, however, may be instanced here in connection with Kronthal's case, which shows how a bruise is liable to produce more extensive deformities in a softened cord of myelitis than in a normal or firm cord.

This cord * came from a man who died in two weeks with an acute idiopathic myelitis, involving the whole dorsal region. The autopsy was made in very hot weather, and the carefully removed softened cord (which was not incised anywhere) was laid on some finely chopped ice in a pail and carried across the city to the laboratory. During its transit the cord had settled down among the ice frag-

* I am indebted for this specimen to Dr. Brothers, then (1886) house physician at Bellevue Hospital.

ments, and the jolting of the latter had contused it, as the subsequent microscopical examination showed, in a most remarkable way, although grossly the cord showed nothing very unusual in its contours. Several of the sections from this cord indicate just as grotesquely the mechanical

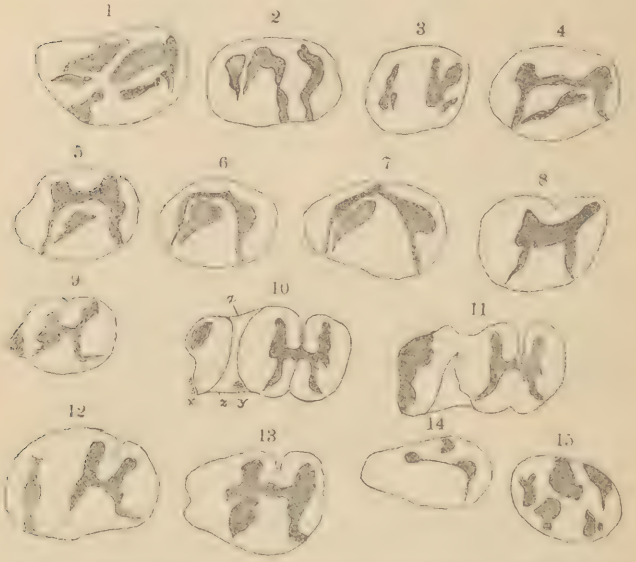

FIG. 7.—Kronthal's first case. Deformities of the cord due partly to acute myelitis and partly to bruising.

origin of the deformities as sections 1, 3, 14, and 15 in Kronthal's first case, Fig. 7. The white matter in the writer's case of myelitis was so much destroyed by the disease that the usual appearances of bruising of the white matter are not as clear as in bruised normal cords.

It is surprising that these writers, who have described malformations or heterotopia of the cord, associated with acute myelitis, should ignore the fact that the myelitis itself, independent of any manipulation or bruising, deforms and destroys the gray matter. I have removed cords of myelitis subjects with extreme caution to satisfy myself of this, and have found that, where mechanical forces may be almost entirely excluded, there may be distortions of the gray matter as the effect of the disease. It may be repeated that most cords in acute destructive myelitis are in such a soft

condition that the slightest touch or handling produces distortions of the cord substances.

Kronthal finally adds a note that the preparation of these heterotopic cords presented great difficulties. The staining was unreliable, and thin sections were hard to obtain. This is all because of the crushed areas of white matter, which get brittle easily and stain somewhat differently from the normal white matter.

No. 9. *Kronthal's Second Case* (29), *1890.*—Kronthal also mistakes a bruise in the cord of an ox for a congenital malformation. The cord came from a butcher's shop. At one point, about one centimetre long, the cord was more voluminous than normal. Kronthal remarks that the rest of the cord was badly damaged by being sawed in two lengthwise, but it never occurs to him that the "anomaly" which he described was also produced by instruments. Sections 1, 2, and 3, Fig. 8, show the appearances of this bruise in the sections. Kronthal also describes the horizontally deflected white fibers usually found in bruises, and queries whether the artificially displaced fibers in section 3 are commissural fibers between the two sets of gray horns, etc. Compare with Fig. 61, Plate XIV, Section V. The author appears to be serious in giving an embryological explanation of the distortions, and thinks that the deflected white fibers pursued a horizontal direction to pass by some obstacle interposed in their path in fœtal times.

Fig. 8.—Kronthal's second case. Artificial deformities in the cord of an ox.

No. 10. *Buchholz's First Case* (30), *1889.*—This writer found an abnormal configuration of the gray matter associated with a corresponding change in the neighboring white matter, in a case of acute delirium. There were no symptoms referable to the supposed malformation. Fig. 9 shows these two sets of changes, which are simply mechanical artifacts. A most characteristic feature of bruising—namely, the deflected bands of horizontal nerve fibers—the author described in detail. (See sections 1 and 2, Fig. 9.) Briefly summed up, the changes consisted in a lengthening out of one or other of the anterior horns, which were in some levels surrounded by horizontal fibers. This condition we have shown to occur quite often when the cord has been cut transversely when fresh and the cord substances have flowed up out of the cut segments. Compare Buchholz's figures—Figs. 5, 6, and 35, Section V.

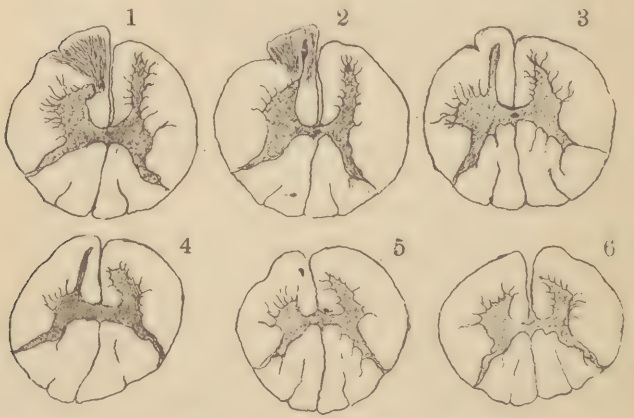

Fig. 9.—Buchholz's first case.

Buchholz considered these misplaced fibers as originally vertical fibers ascended from lower regions, which, after turning out horizontally, continued again in a vertical course. A great number of small hæmorrhages were also found in this case. While these were probably associated with the acute delirium during life, they may have also been caused by the bruising. (Compare Fig. 62, Section V.)

No. 11. *Buchholz's Second Case* (31), *1890.*—A second case of supposed developmental anomalies of the cord is re-

ported by the same author, in a case of dementia paralytica, with no symptoms referable to the deformity. Fig. 10 shows the character and distribution of these supposed malformations. There was descending degeneration in the crossed pyramidal tracts. The deformities, especially the

FIG. 10.—Buchholz's second case.

deflected columns of white matter which are noted in the text, indicate a mechanical origin of the distortions. The author indorses the opinion of Schultze, Fürstner, and Zacher that such supposed developmental anomalies may form an anatomical substratum for the neuropathic disposition.

No. 12. *Jacobssohn's Case* (32), *1891.*—Jacobssohn's case is another bruised cord of acute myelitis, like Kronthal's, in which distortions due to the disease or, more probably, to mechanical violence, are described minutely, and supposed to be malformations of congenital origin. (See Fig. 11.) When the cord has been so thoroughly knocked to pieces by bruising as shown in Fig. 11, sections 1, 2, and 3, it is difficult to understand how the appearances can be considered as malformations and the case be published as one of heterotopia. Surely, a cord in the condition shown in sections 1 to 3 could not perform its functions. Jacobssohn also describes a partial doubling of the cord in the lumbar region. Grossly, the cord appeared normal in its contour, except that it was more voluminous than usual in the lumbar region.

No. 13. *Brasch's Case* (33), *1891.*—Brasch describes in detail mechanical distortions, as heterotopia, in the cord

from a case of syphilis of the central nervous system simulating the symptoms of tabes. (See Fig. 12.)

Fig. 11.—Jacobssohn's case. Bruise artifacts associated with acute myelitis.

Remarks.—The cord appears to have been bruised over a considerable territory; a good part of the dorsal region and the lower end of the cervical region show these mechanical artifacts. In the eighth dorsal segment a column of white matter, deflected mechanically, has been thrust into the gray matter, and disfigures the left anterior horn. In the seventh dorsal segment the same bundle of disarranged white fibers has produced a still greater effect on the configuration of the gray matter, and separates the distorted and fused anterior horns from the posterior horns. (See Fig. 12, section 2.) It is interesting to note that the author describes the concentric arrangement of horizontal fibers about the displaced white column shown in Fig. 12. This occurs occasionally in bruises. (See Fig. 15, Section V.) In the cervical region, the splits and clefts described by the author indicate a mechanical origin. Many of the sections showed defects of the cord substances characteristic of severe bruising.

Minute structural changes are also described in the bruise dregions, such as irregular degenerations or scleroses

of the white matter, which in places were very extensive. Although these structural changes were probably largely caused by pathological processes, or the syphilitic element, one can not feel sure whether the author distinguished rig-

Fig. 12.—Brasch's case.

idly to what extent these minute structural changes were due to the bruising, and to what extent they were actually caused by pathological processes.

We have repeatedly shown that bruises, in addition to the production of topographical changes, may also produce minute structural alterations which resemble closely degenerations and sclerosis of the cord.

When a diseased cord, especially a cord with irregular degenerations or sclerotic patches, is bruised, it becomes a very difficult, if not an undeterminable, question in some cases to tell how much of the minute structural changes are due to disease processes and how much are simply artifacts.

At the conclusion of this paper, Brasch discusses the question of the association of the neuropathic disposition with these malformations, reviews other cases, and intimates that these artifacts make the cord prone to, or less resistant to disease, etc.

No. 14. *Feist's First Case* (34), *1891.*—This case relates to a man having typical paretic dementia, who died of sepsis three weeks after a fracture of the arm; there

Fig. 13.—Feist's first case.

were, of course, no symptoms of the alleged congenital malformations of the cord. Feist describes the various topographical and structural changes in the cord due to bruising with much thoroughness and detail. Wholly unaware of the artificial and mechanical origin of these changes, he considers them preformed, and describes them as anomalies in the course of the fibers of the white matter and partial doubling of the spinal cord. Feist's drawings (reproduced in part here in Figs. 13 to 20 inclusive) are very conscientious, and are by far the best in the literature, because they are on a large scale and show the details of the changes in the white matter so characteristic of bruising. Except Feist, nearly all of the writers who have mistaken bruises of the cord for congenital malformations have paid most attention to the altered topography of the gray matter, and have

neglected, both in drawings and text, the details of the changes in the white matter.

FIG 14.—Feist's first case.

The anomalous bundles and sets of fibers in the white matter running in abnormal directions, described and illustrated so accurately by this writer, are simply the strands

FIG. 15.—Feist's first case.

of white matter mechanically deflected by the bruising, of which so many examples are shown and described in Sections IV and V.

In the first part of the paper, bundles of artificially displaced white matter passing out horizontally to the periphery of the cord between the two anterior horns (see Figs. 13, 16, and 17) are described as aberrant or anomalous bundles. (Compare with Figs. 6, 35, and 36, Sections IV and V.) Feist at length describes what he supposes to be a curious malformation, consisting in horizontal and vertical

FIG. 16.—Feist's first case.

bundles of white matter inside of the central canal (open in this case), and inside of the gray commissure (Figs. 14, 16, and 17). In one place, Fig. 14, an "aberrant" bundle passed through a portion of the gray commissure. These supposed aberrant bundles inclosed in the gray matter are simply portions of the white matter which had been driven by the force of the bruise into the perivascular spaces of

the sulco-commissural arteries in Figs. 15 and 17, and into the adjoining open central canal in Fig. 16. The sulco-commissural perivascular spaces communicate directly with

FIG. 17.—Feist's first case.

the cavity of the anterior fissure, and crushed fibers which had been thrust out of Türck's columns into the anterior fissure (as was the case here, see Fig. 13) would naturally be squeezed into these perivascular orifices opening into the fissure, and might also pass into the neighboring open central canal. In some places the crushed fibers have not passed from the anterior fissure to the perivascular spaces

in this way, but have reached these spaces by directly breaking through and rupturing the anterior white and portions of the gray commissure, as in Fig. 14.

The forcible pressure of the bruise exerted by these aberrant portions of the white matter is also shown in the manner in which they have squeezed and distorted the central canal in Figs. 14 and 15.

The fact that the sulco-commissural apertures communicate by vertical anastomoses (Adamkiewicz *) makes it not at all remarkable that Feist found the white bundles artificially inclosed in the gray commissure for some little vertical distance. For if the bruised white matter entered these commissural spaces at only one level, it could have been dispersed up or down along these vertical anastomosing sinuses.

After describing these supposed aberrant bundles, the author describes the gross and microscopical appearance of

Fig. 18.—Feist's first case.

a partial doubling of the cord in the same case. Feist considers this doubling of the cord as a congenital malformation, but he is clearly wrong about the matter, and the doubling is nothing more or less than the result of an autopsy bruise

* Adamkiewicz. *Die Blutgefässe des menschlichen Rückenmarks*, Part II.

of the cord. The microscopical description may be dispensed with. His drawings, copied in Figs. 18, 19, and 20, are substituted for it, and they show plainly enough what has happened to the cord in the way of bruising. It seems advisable, though, to repeat the gross appearances with some detail to show how observers continue to bruise the spinal cord at the autopsy, and describe the results as suggesting

FIG. 19.—Feist's first case.

tumors, new growths, or structural peculiarities, without appreciating the artificial nature of the changes. Feist describes the appearances as follows:

"Between the exits of the twelfth dorsal and second lumbar nerves a flattened tumor, eighteen millimetres long and eight millimetres wide, lay upon the anterior surface of the cord. The pia mater surrounded the cord up to its junction with the base of the tumor, but did not cover the tumor itself, and I supposed that it was a new growth of the pia mater (gumma or the like) which perhaps had involved the spinal cord. At its proximal end the tumor was firmly adherent to the spinal cord, and presented a compact structure with a lustrous yellowish white surface. Distally, the tumor was cleft by several longitudinal splits and was more loosely attached to the pia or cord."

This is a fairly good gross description of a spinal-cord bruise, and the appearances were probably not unlike those in Fig. 17, Plate V. It is somewhat unusual that no deficiency was noticed in the cord, due to the extrusion of the cord substances which produced the tumor-like mass.

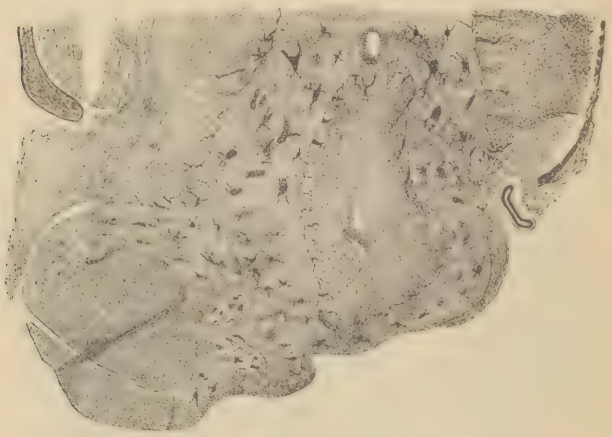

FIG. 20.—Feist's first case.

Figs. 18, 19, and 20 show the microscopical appearances of this bruised region. The drawings are very faithful in detailing the changes in the white matter, and give a good idea of how irregular these artificial doublings of the cord generally are. There is a helter-skelter fragmentary arrangement of extra portions of gray and white matter, due to the fusing together of different portions of the cord from consecutive levels.

Besides detailing the topographical changes due to the bruising, Feist also gives a commendable description of the minute structural changes in the nerve fibers in the bruised regions. He notes the changes in the myelin and the axis cylinder in the crushed fibers, and the aggregation of certain of the crushed fibers, so that with moderately low powers they looked like finely granular masses. The behavior of these structural artifacts with different staining reagents is also described.

Feist describes very well these minute structural artifacts due to bruising, but he considered these artificial changes

in the deflected white bundles as due to a pre-existing degenerative process in the nerve fibers. Bands of neuroglia, which had been squeezed together mechanically in these deflected bundles, are interpreted as neuroglia thickenings and hyperplasias, going hand in hand with the degenerative changes in the nerve fibers.

The finer structural changes in the white and gray matter induced by bruises has probably not infrequently been ascribed to pathological processes by other writers, especially in the literature of cord malformations; but most of the writers describe the artifacts in the white matter so loosely that one can not be positive whether they are describing artifacts or the results of the particular disease for which the cord was removed. Feist's position, however, in this matter of mistaking the minute structural artifacts of bruising for the effects of pathological processes is quite unmistakable. Although his conclusions and interpretation of the cord changes are all wrong, yet compared with the other papers on this subject, Feist's paper is the best in the literature.

FIG. 21.—Campbell and Turner's case.

No. 15. *Case of Campbell and Turner* (35), *1891.*—These writers describe heterotopia of the cord substances in a case of ordinary transverse myelitis in the lower dorsal region. The authors refer to the comparative rarity of the heterotopic condition and point out the

liability to myelitis which this faulty structural arrangement of the cord possessed. The clinical history is interesting. For some considerable time (a year or more) before the myelitis occurred there was apparently some cord lesion which induced constantly recurring trophic changes in the skin, nails, and nearly all of the joints. The myelitis was of the subacute variety and came on gradually.

Remarks.—Notwithstanding the accurate description of the changes in the cord—the writers refrain from making any positive conclusions about the heterotopic condition—this case appears to be quite similar to those of Kronthal and Jacobssohn, in which the destructive effects of the myelitis and the difficulty of handling such cords without bruising them are responsible for the deformities (see Fig. 21). It should be noted that the distribution of the deformities, extending from the sixth to the twelfth dorsal segments, occupied the regions of the cord involved by the myelitis.

No. 16. *Turner's Case* (36), *1891.*—At a meeting of the London Pathological Society, Turner referred to some sections of a rabbit's spinal cord which showed a heterotopic condition. There was an accessory anterior horn, with two central canals, and three anterior nerve roots.

This specimen is described so briefly without drawings that it is impossible to determine anything at all positive about the nature of the deformity. While not denying that there is a possibility that these appearances indicate a real malformation, I am more inclined to believe that the deformities are bruise artifacts. An extra anterior horn is frequently thrust to another level by bruising, and it is possible for a nerve root to be shifted along with the transposed horn. (Compare with Fig. 39, Section V.)

No. 17. *Tooth's Case* (37), *1891. Remarks.*—Tooth records deformities associated with acute and chronic inflammatory processes in a spinal cord(removed by some one else) from a man aged twenty-four years who had dementia paralytica. The deformities occupied the whole of the dorsal and portions of the lumbar and sacral regions of the cord (see Fig. 22). Although not explicitly recognizing the artificial origin of the deformities, the author in his excellent report of the microscopical appearances seems to protest against accept-

ing the distortions as malformations, for some of the sections are so described as to intimate an appreciation of the

FIG. 22.—Tooth's case.

results of mechanical forces. For instance, the gray matter in places is described as having been broken into pieces or moved away from its proper position, and portions of the gray matter looked as if they had been chipped off or dislocated from the gray segments. Tooth also recognizes one or two features difficult to reconcile with the appearances of actual malformations of the spinal cord. For example, the author points out in one place that although the anterior horn had been moved away from its proper position, the entrance fascicles of the corresponding anterior root remained in their appropriate situation disconnected from the shifted anterior horn. At any rate, Dr. Tooth clearly expresses his suspicions of the heterotopic changes as a primary condition in his final paragraph appended below.

"The question now arises whether the appearances in heterotopia be secondary to inflammation or whether they existed before as a developmental abnormity, which has since become the seat of acute or chronic inflammation. An inquiry into the diseases to which the recorded cases succumbed does not throw much light upon the subject, except to show that they most of them died of some chronic nervous disease, several from general paralysis. The examination of the cord in the case which is the subject of this communication leads one rather to the conclusion that a widely spread inflammation of the gray matter is at the bottom of the affection."

The deformities in this case were no doubt caused, in part, by the associated lesions, but it is very likely that the bruising or manipulation of the diseased cord was more largely responsible for their presence.

No. 18. *Chiari's Case* (38), *1891.*—This case consisted in a partial doubling of the cord in a very young child having spina bifida, and is very briefly alluded to. In a portion of the lumbar region there were two normal anterior horns, but four well-formed posterior horns which were properly connected with their nerve roots. The increased substance of the posterior white columns was uniformly medullated.

The absence of drawings and definite details of this deformity renders it impossible to draw any conclusions as to the real nature of the changes. The deformity may

possibly have been of congenital origin, or it may have been produced readily enough by mechanical means; so it is difficult to know whether to class Chiari's case among the true cases or among the erroneous cases of malformations of the cord. (The case would seem to me to look more like a bruise than a malformation.)

No. 19. *Gyurman's Case* (39), *1892.*—Gyurman, in examination of a case of syringomyelia, describes an additional anterior horn surrounded by extra white matter, fused with the cord on its right lateral aspect. Gyurman describes this as heterotopia, and believes it to be of congenital origin. I doubt the congenital origin of this sup-

FIG. 23.—Gyurman's case.

posed heterotopic condition, and have selected one of Gyurman's drawings (see Fig. 23) as indicating a bruise of the cord.

No. 20. *Feist's Second Case* (40), *1892.*—In this case are described with the same thoroughness as in the first instance malformations of the gray matter, and "aberrant" bundles of white matter, in the spinal cord of a man aged thirty-eight, who had had fatal general paresis for two years and a half. The case was not complicated by cord disease, except that there was a degeneration of the columns of Goll and a trifling marginal sclerosis. The appended drawings (see Fig. 24), selected from the author's plates, show very faithfully the artificial character of the supposed heterotopic condition.

No. 21. *Köppen's Cases* (41), *1892.*—Köppen cites two cases of "acute formation of cavities in the spinal cord"—

Fig. 24.—Feist's second case.

one which showed heterotopia of the gray matter. These are reviewed here not to class them positively as erroneous cases, but to suggest that the artificial causes which may have produced both the cavities and the heterotopia ought to be excluded before accepting the ante-mortem origin of the changes.

In the first case there was an atrophic paralysis of both legs and a trifling disturbance of sensibility and of the bladder. Shortly before death right ptosis and contraction of the right pupil occurred. The autopsy revealed involvement of the lumbar and to some extent of the sacral plexuses by sarcomatous masses either side of the vertebral column. The spinal cord showed an ascending degeneration of the posterior columns, and in the cervical region a cavity situated in the middle of the gray matter. *The cavity had no lining wall, and was not connected with the central canal.* Larger and smaller hæmorrhages were scattered about in the surrounding gray matter. There were a few changed cells and nerve fibers, but no cell proliferation.

The second case related to a man who had had rather sudden loss of power, and spasms in the legs, trembling in the hands, extensive bronchitis, and progressive insensibility. The cord was normal up to the cervical enlargement. In the upper cervical enlargement *the gray matter showed a congenital anomaly, consisting in the presence of two anterior horns on one side. The lower level of this anomaly contained a torn or ragged cavity in the deformed gray matter.* The anomalous gray matter also showed numerous small hæmorrhages and diffuse infiltrations of red blood cells, but no signs of inflammation.

Remarks.—The author regarded these cavities of acute origin, and believed them due to a hæmorrhagic œdematous softening (?), induced to considerable extent by the cachectic condition of the patients. In case 2 the circulatory disturbances were thought to have been augmented by the heterotopic condition.

I am unable to interpret the "acute cavity formations" of Köppen in any other way than as artifacts. Several very important factors seem to show that there is no proof whatsoever of the cavities having been formed by pathological processes. The cavities had no lining walls or sur-

rounding evidences of inflammation or necrosis, and were not associated with disintegrating neurogliar tumors or hyperplasias. Nor do the symptoms indicate the cavities or correspond very well with their location. In the first case the symptoms are perfectly well explained by the localized lesions of the nerve plexuses. In the second case, also, the correspondence of the indefinite symptoms in the arms with the character and situation of the cavity is not very impressive. Furthermore, in the second case the cavity is most distinctly associated with a palpable bruise-artifact in the duplicated anterior horns on one side of the cord.

Taken altogether, these cavities seem to be quite identical with the holes in the gray matter produced by mechanical disintegration or rarefaction (see Drummond's case), or with the other ragged-walled splits and cavities caused by bruises described in Sections IV and V. The cavities in the bruised spinal cord—Section V (Fig. 15)—should be referred to in connection with these cases of Köppen. The hæmorrhages in the gray matter might also have had an artificial origin.*

This *résumé* includes nearly all of the mistaken cases of artifacts of the spinal cord, and it will be seen that the appearances in the drawings are quite similar to the artifacts shown in the plates of Sections IV and V.

Section VII.

THE DIAGNOSIS OF SPINAL-CORD BRUISES.—GENERAL REMARKS.

When the true cases of heterotopia are compared with the erroneous instances, or the results produced by bruising the cord, the contrast is quite striking. In all of the true cases of heterotopia of the gray matter the gray crescents throughout the cord are not misshapen, nor do they show any deficiencies; on the contrary, they are perfectly well developed and have their typical contour and symmetry. The white matter is also undisturbed; the volume and course of

* Köppen's cases are meagerly described in a society report (*Berliner Gesellschaft für Psychiatrie und Nervenkrankheiten*, July 11, 1892), which may express his views incompletely.

its fibers are perfectly regular. There is simply a redundant bit of gray matter (not corresponding to loss of substance elsewhere in the gray horns as in the artificial cases) which was left behind during the development of the gray horns. The rest of the cord has gone on and developed perfectly in every respect. The same may be said of the abnormal tracts of white matter in the medulla; the surrounding structures are perfectly well developed, and the congenitally misplaced bundles of fibers performing their functions properly, reach their prescribed destinations, but have simply strayed away from their customary paths.

This is what would be expected in actual cases of heterotopia, and is in accordance with similar malformations in other organs. It is also perfectly natural in a developmental error of this kind for a few ganglion cells to remain exiled from the parental gray horns, in the residual fragment, as described in one or two of the cases. It will be noticed also, in the true cases of heterotopia of the gray matter, in accordance with the tendency for a complex organ to show preferably developmental aberrations in its lowly organized elements rather than in its more highly organized structure, that the inferiorly constituted tissue of the spinal gray matter—the Rolandic substance—has been most frequently the source of the malformation.

In the artificial deformities there is an entirely different state of affairs. The gray horns are misshapen, and extra or dislocated gray portions can be traced by serial sections to corresponding deficient areas in the gray horns above or below the plane of the section. The white matter is almost always disturbed. The distorted contours of the gray horns and the extra gray portions are generally surrounded or accompanied by mechanically deflected tracts of white matter. If not deflected in various directions in this way, the neighboring white columns are squeezed or crushed together. If there are extra portions of white matter in the section, it is more difficult to trace their source, but the nerve fibers show invariably the effects of mechanical forces. Artificial deformities generally involve a considerable extent of the cord as compared with the actual heterotopic condition, which, as shown by the cases thus far, occupies an exceedingly limited space in the organ. Thus the artificial de-

formities have distinctive features which enable them to be accurately distinguished from actual cases of malformations or heterotopia.

Some exceptional cases of bruises have less characteristic traits to distinguish them from actual cases of heterotopia or the results of disease.

In rare instances of spinal-cord bruises a tiny bit of gray matter may be displaced some little distance to a nearly normal level with but comparatively little disturbance of the white matter, and it is possible that one or two sections through the upper level will show a nearly perfect contour of the gray segments and hardly any changes in the white matter surrounding the displaced gray fragment. Then the appearances may resemble a true case of heterotopia very closely such as in the first two cases of Pick. Thus, no matter how much a displaced gray fragment may simulate actual heterotopia, serial sections should be made some little distance above and below the suspected level to eliminate artifacts of the white matter and a deficiency in the gray horns corresponding to the misplaced fragment before pronouncing the case one of true heterotopia.

In bruises, as a rule, deflections of the white matter and deformities of the gray matter go hand in hand and are in direct ratio to each other—that is, the more extensive the deformities of the gray matter the more extensive are the mechanical artifacts of the white matter. There are, however, notable exceptions to this rule. A single limited and quite small column of deflected white matter may pass across or indent the gray horns and distort them considerably, and on the other hand there may be quite extensive deflection of the marginal white matter, with but little if any disturbance of the gray horns. In one or two instances of experimental bruises the whole section was perfectly normal in the shape and arrangement of the cord substances, with the exception of a single circumscribed column of deflected white matter.

The influence which actual heterotopic conditions exert over the cord in rendering it prone to disease must be quite inconsiderable. There seems to be no reason why these tiny heterotopic fragments of such infrequent occurrence should not remain indefinitely buried without

being resurrected into a significant lesion. Of course, in accordance with the now established hypothesis of Cohnheim, we might regard these tiny fœtal relics as a nidus for the development of tumors. But tumors of the type of the Rolandic tissue are seldom if ever observed; yet, as all of the neural tissues develop from the same embryonic layer, it is possible, though not very probable, for any of the tumors of the glioma group to originate in such heterotopic fragments, for they are generally so poorly supplied with cells and consist so largely of the Rolandic basement substance.

It might seem appropriate to speak further of the neuropathic disposition so much associated with this theme. But it concerns us here only to show that a good deal of unwarrantable responsibility in connection with cord malformations has been shouldered upon the neuropathic disposition. It is finally to be hoped that the recently accumulated and tangible knowledge of the causes of pathological processes may release neural diseases, especially the organic ones, from the speculative dominion of the neuropathic disposition.

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PLATES IV–VI.

Normal spinal cords in the fresh condition, showing effects of the more severe bruises which may occur in the technique of removal at the autopsy. These artificial appearances are quite generally mistaken for malformations, tumors, or other lesions of the spinal cord, but are produced unawares at the autopsy by bruising the cord with instruments (chisel, mallet, osteotome) or fragments of bone.

PLATE IV.

FIG. 16.—Shows the very perfect simulation of the fresh gross appearances of acute myelitis by an accidental autopsy bruise of the lower dorsal region of the spinal cord of a horse. The bruise inflicted by autopsy instruments has produced a soft, pulpy, fusiform swelling of the cord with a trifle of flattening. This drawing was originally made two years in advance of these researches, in 1887, to show what was then supposed by the writer to be a focus of acute myelitis with softening in a horse having partial paraplegia. The bruise inflicted at the autopsy by the author was wholly unappreciated until the case was latterly re-examined in conjunction with experimental cord bruises, when the characteristic artificial changes of bruising were understood and the cord tissues found to be perfectly normal.

PLATE V.

Showing results of attempts to produce artificial duplication of the human spinal cord. Each of these two portions of the cord, after careful removal, was bruised by bimanual pinching with the fingers on either side of a given point. In this way portions of the cord were transported or telescoped to other levels.

FIG. 17.—The displacement of the cord substance by the bruise has produced a hard, lumpy swelling in the upper portion of the cord, while below a portion of the cord has been thrust out laterally through the pia mater. (For microscopical appearances, see Plate XV, Fig. 62.)

FIG. 18.—A portion of white matter from a contiguous level has been extended over the anterior surface of the cord. The deficiency corresponding to the extruded portion was not pronounced in this case. (For microscopical appearances, see Plate XV, Fig. 63.)

PLATE VI.

Human spinal cords severely bruised by careless technique in removal at the autopsy.

FIG. 19.—The cord has been struck so forcibly with the chisel that a portion of it has been forced out through the dura mater. (For microscopic appearances, see Plate XV, Figs. 64, 65.)

FIG. 20.—The cord was injured by driving down fragments of the vertebral arches into the vertebral canal with the chisel and mallet. The deficient indented region of the cord indicating the source of the lateral extended portion—a rather characteristic feature of severe bruises—is to be noted.

NOTE.—*The figures in these plates should be numbered 16, 17, 18, 19, 20, as in the descriptions, in order to make them conform to the references in the body of the article.*

Plate I.

Fig. 1

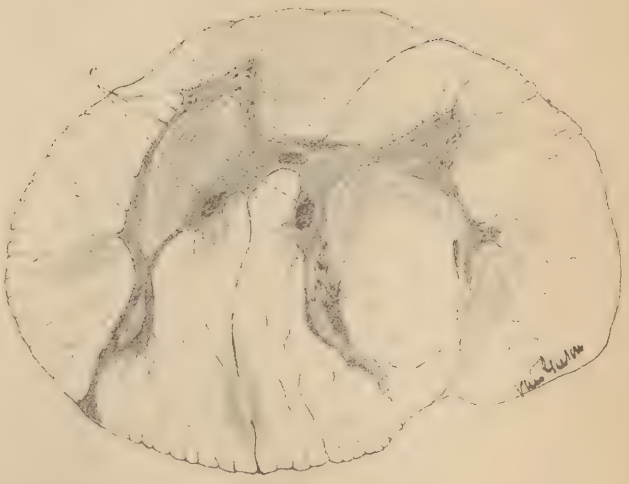


Fig. 2

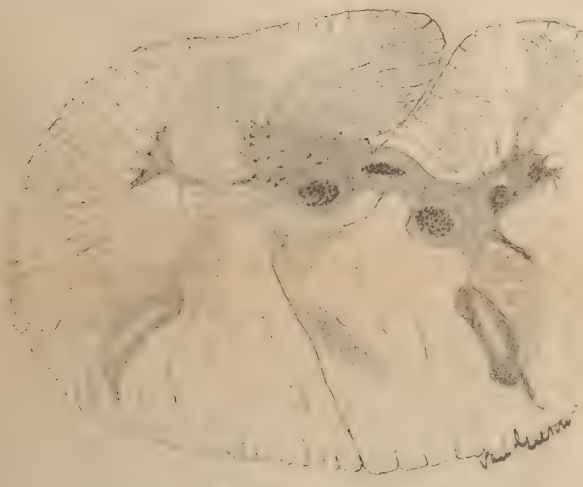


Fig. 3

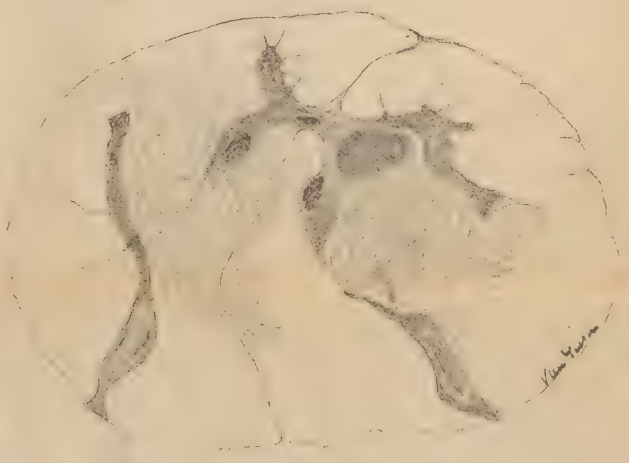


Fig. 4

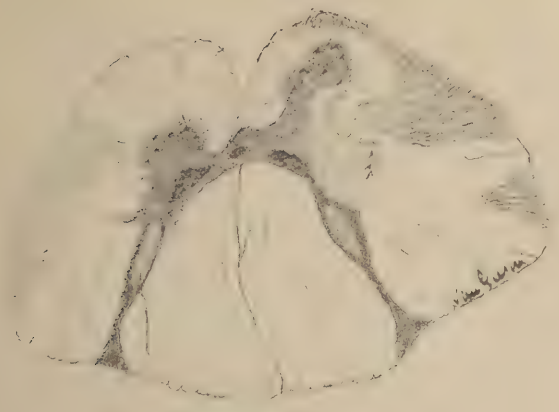


Fig. 5

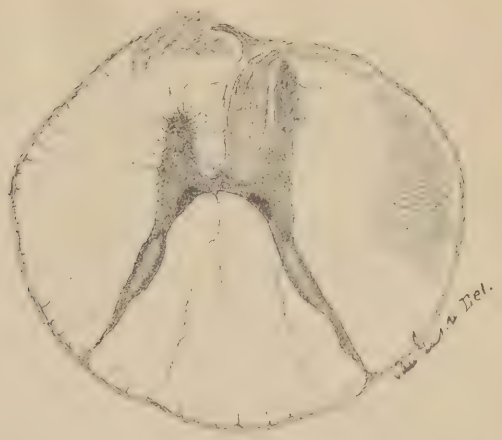


Fig. 6

Sections from a case with obscure spinal symptoms bruised unawares at the autopsy.

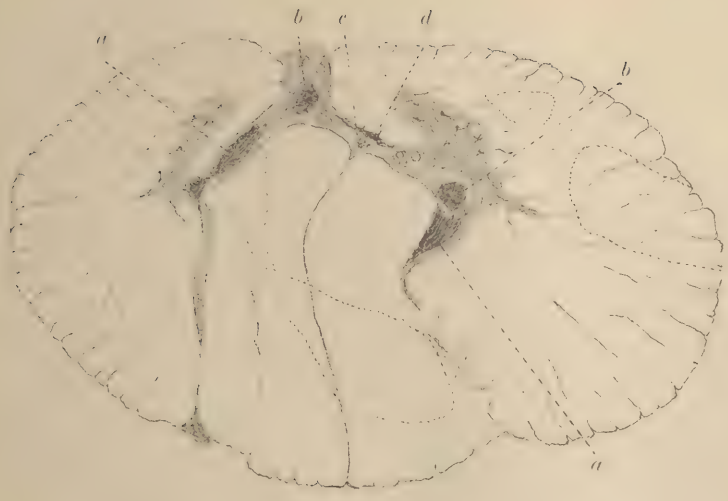


Fig. 7

Fig. 8

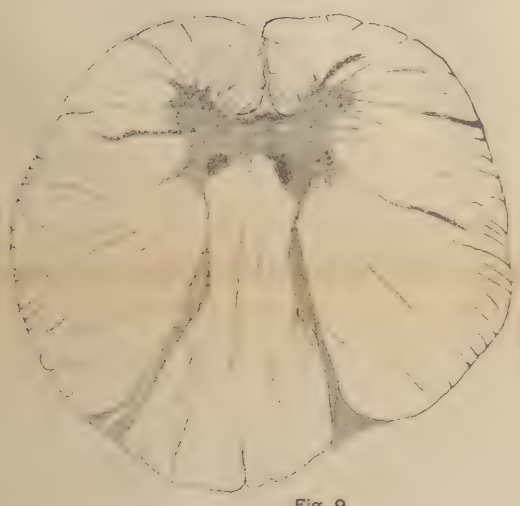


Fig. 9

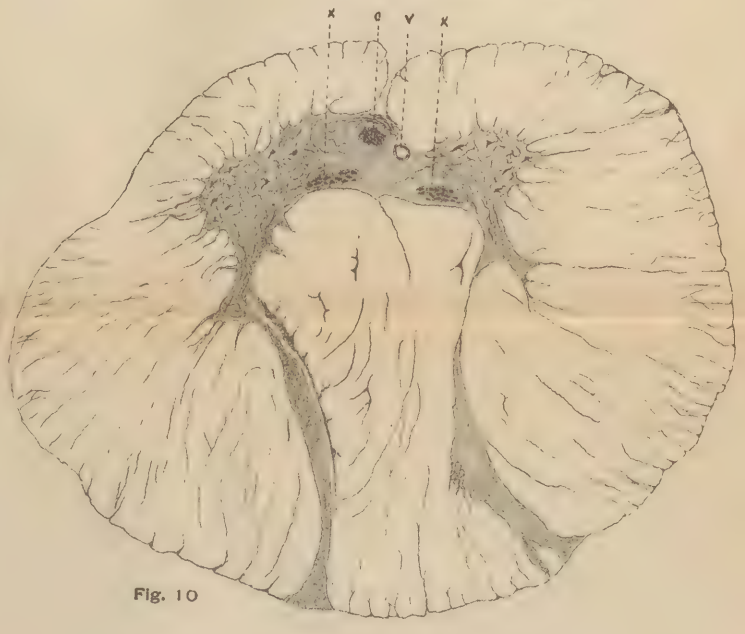


Fig. 10

Fig. 12

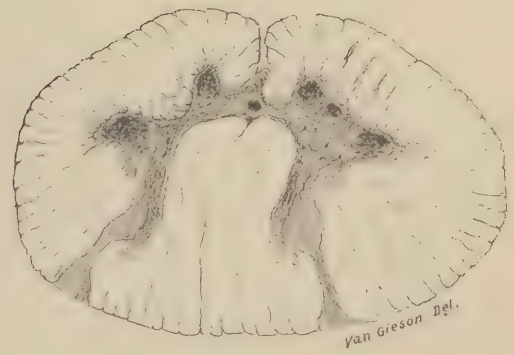


Fig. 11

Sections from various cords, unintentionally bruised at the autopsy, found in the laboratory collection of spinal cords.

Plate III.

Fig. 13

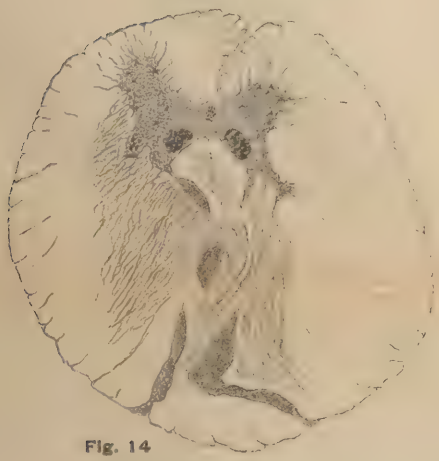


Fig. 14

Fig. 15

Sections from the cord in a case of chorea, bruised unawares at the autopsy.

Plate VII.

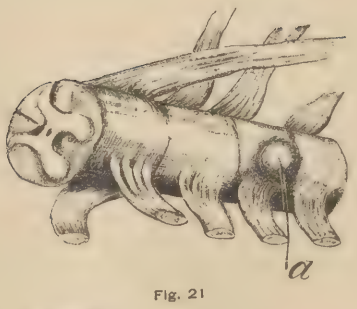


Fig. 21

Fig. 22

Fig. 23

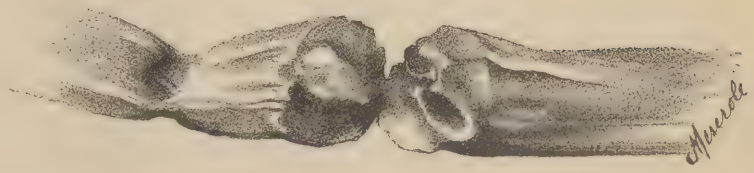


Fig. 24

Gross appearances of bruises of the spinal cord.

DR. IRA VAN GIESON'S ARTICLE.
A STUDY OF ARTIFACTS OF THE CENTRAL NERVOUS SYSTEM.—HETEROTOPIA OF THE SPINAL CORD.

Plate VIII.

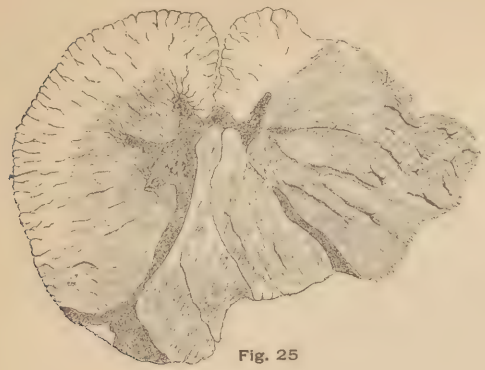


Fig. 25

Fig. 26

Fig. 27

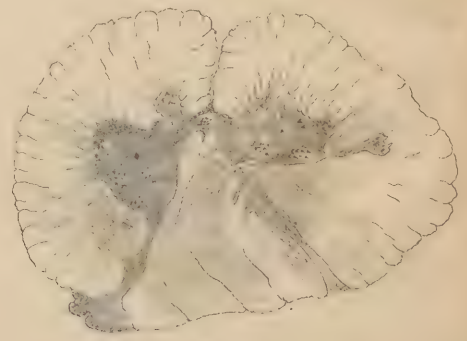


Fig. 28

Fig. 29

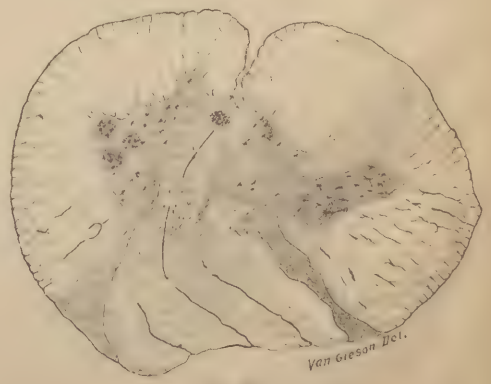


Fig. 30

Sections from an experimental bruise of the spinal cord in the lower cervical region.

Plate IX.

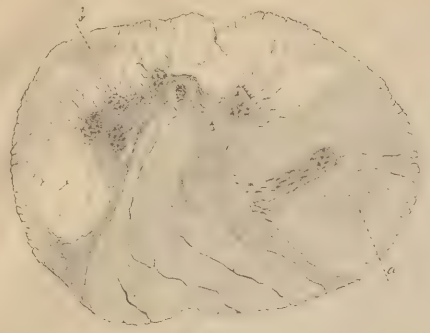


Fig. 31

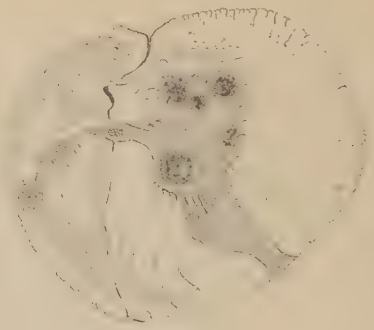


Fig. 32

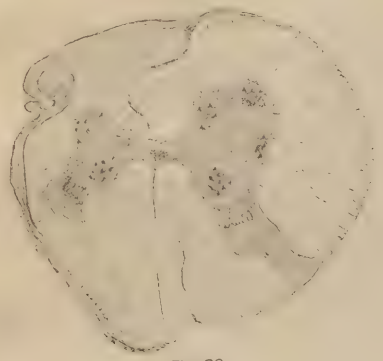
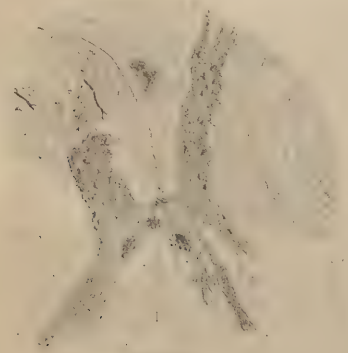


Fig. 33

Fig. 34



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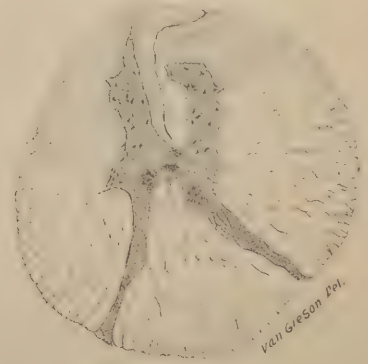


Fig. 36

Microscopical appearances of experimental bruises of the spinal cord.

Plate X.

Fig. 37

Fig. 38

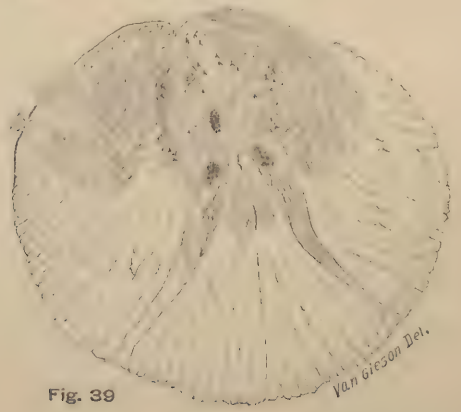


Fig. 39

Plate XI.

Fig. 40

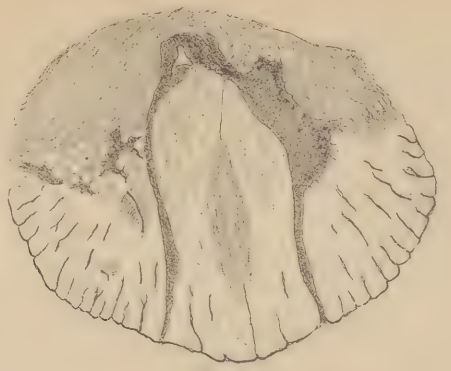
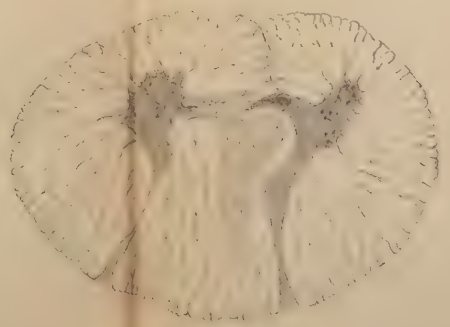


Fig. 41

Fig. 42

Fig. 43



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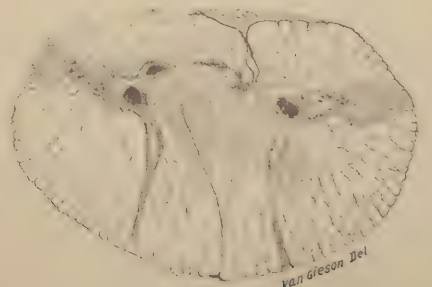

Fig. 45

Microscopical appearances of experimental bruises of the spinal cord (continued).

Plate XII.

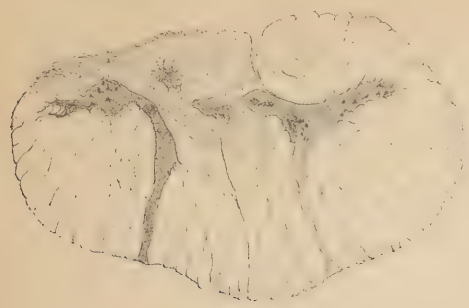

Fig. 46

Fig. 47

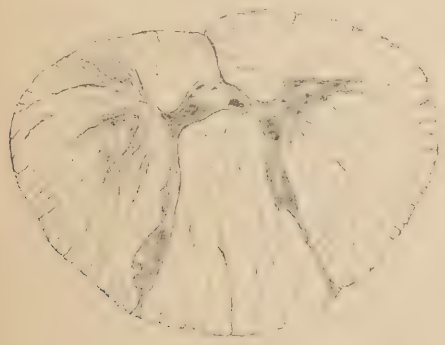

Fig. 49

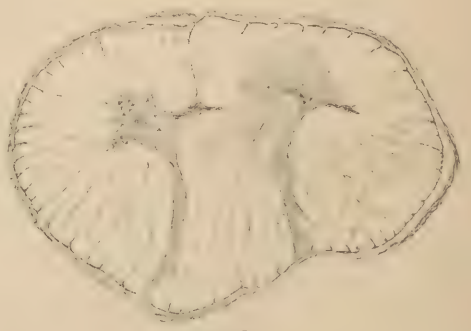

Fig. 48

Fig. 50

Fig. 51

Microscopical appearances of experimental bruises of the spinal cord (continued).

Plate XIII.

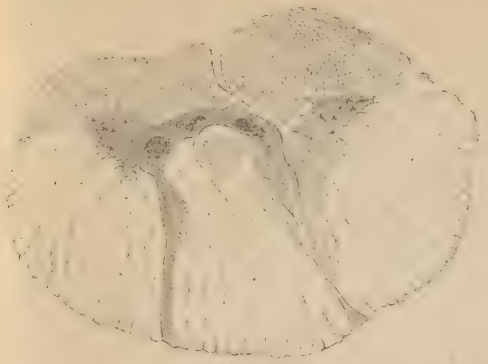

Fig. 52

Fig. 54

Fig. 53

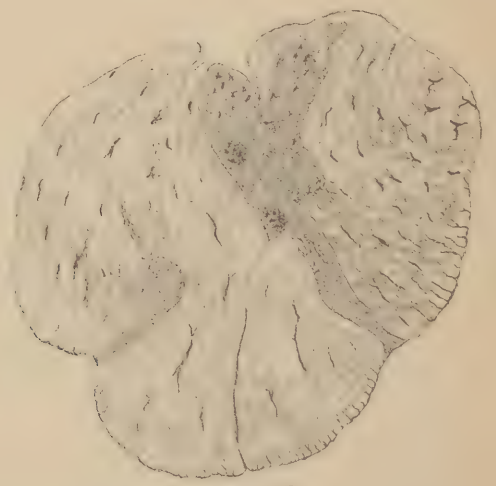

Fig. 55

Fig. 56

Sections from experimental bruises of the spinal cord (continued).

Plate XIV.

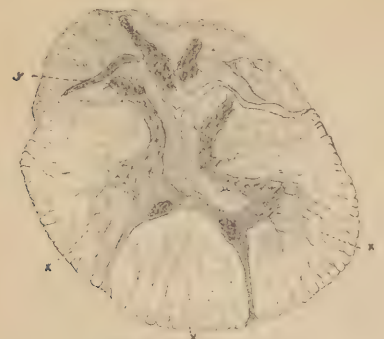

Fig. 57

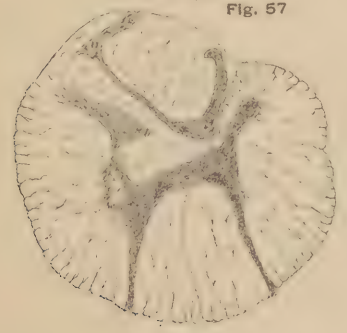

Fig. 60

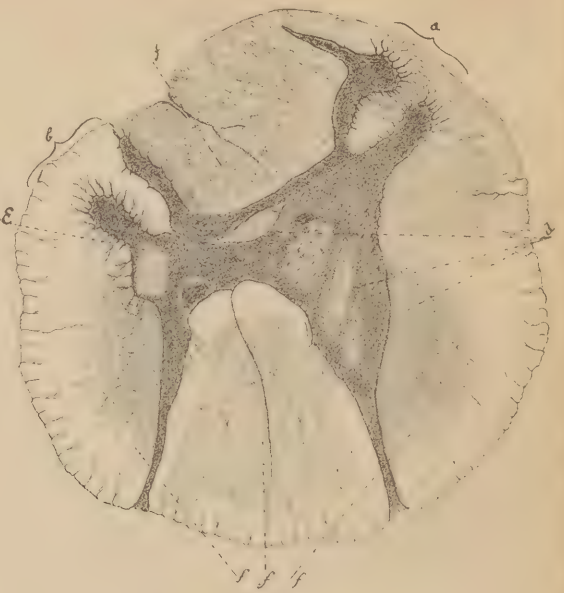

Fig. 61

Fig. 58

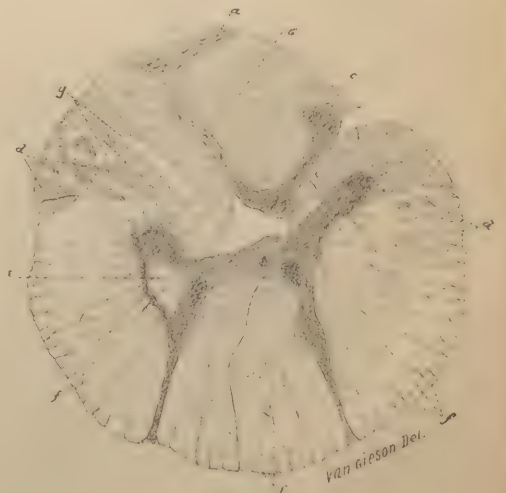

Fig. 59

Artificial duplication of the spinal cord resulting from a bruise.

Plate XV.

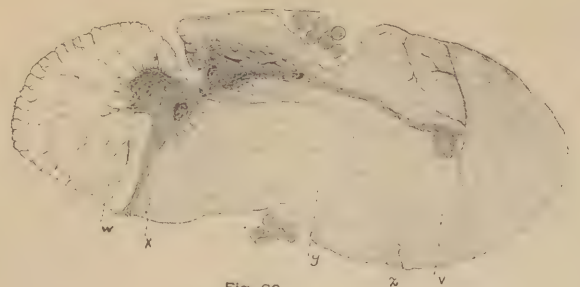

Fig. 62

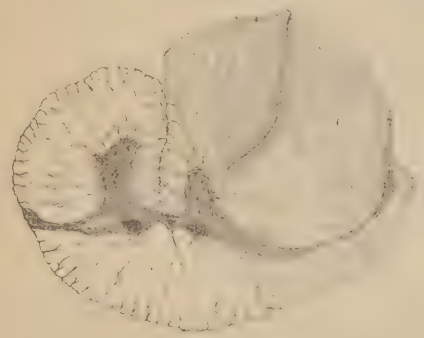

Fig. 63

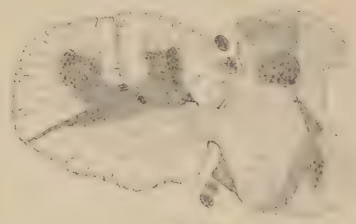

Fig. 66

Fig. 64

Fig. 65

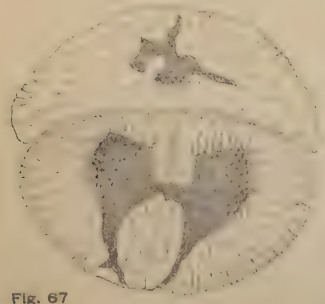

Fig. 67

Fig. 68

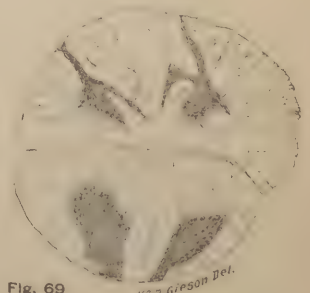

Fig. 69

Sections from several forms of artificial duplications of the spinal cord produced by bruises.

Plate XVI.

Fig. 70

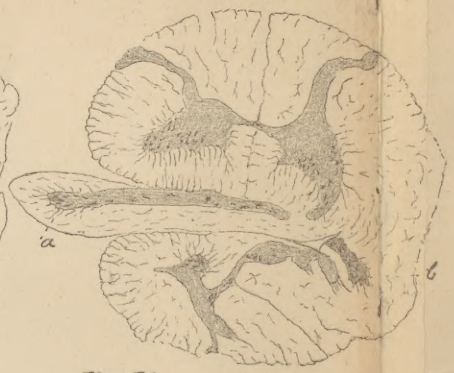

Fig. 71

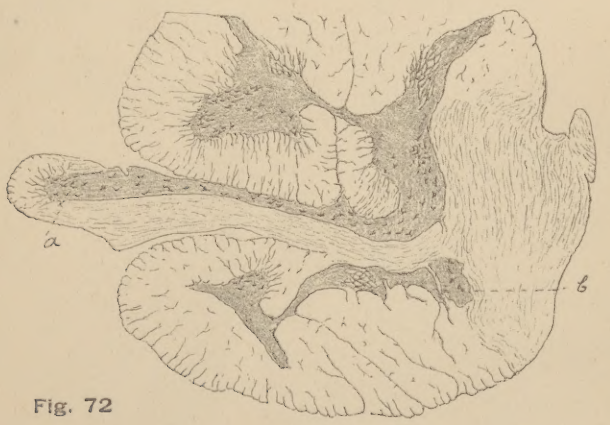

Fig. 72

Fig. 73

Fig. 74

Fig. 75

Artificial duplication of the spinal cord due to bruising.

Plate XVII.

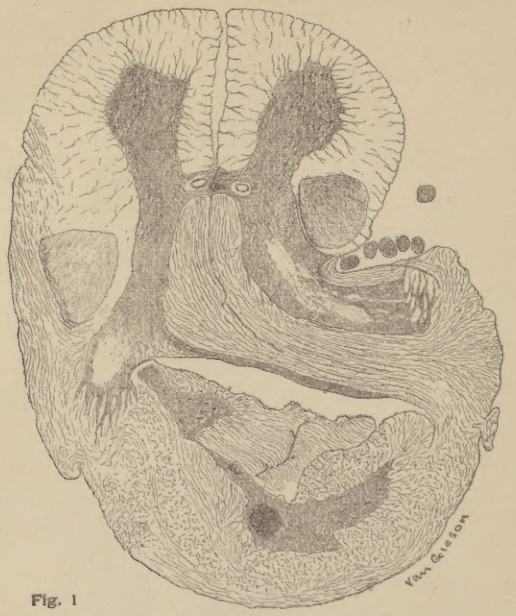

Fig. 1

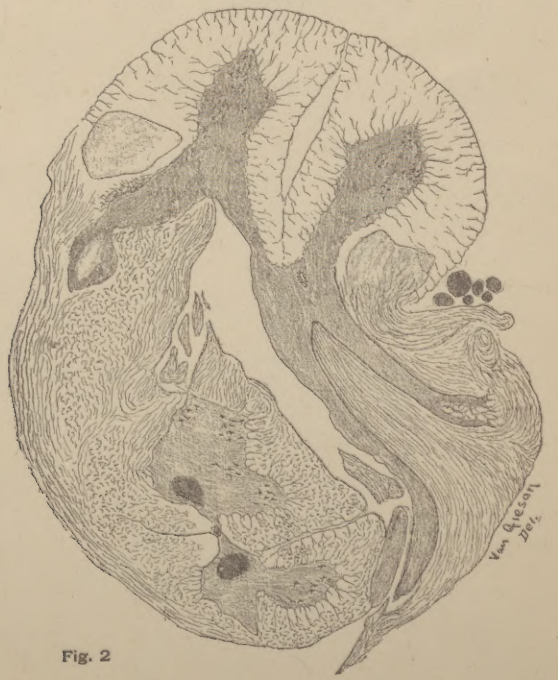

Fig. 2

Seguin's case. Duplication of the cord from an unappreciated autopsy bruise.